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HEREDITARY ENCHONDRAL DYSOSTOSIS

Twelve cases in three generations mainly with peripheral location

by

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Hereditary bone deformities have often been studied by roentgenography. RIBBING, MÜLLER, BRAILSFORD, ROMANUS, HERMODSSON, and others have published reports on hereditary changes mainly located in the epiphyses. Unfortunately, full agreement has not yet been reached regarding the terminology for these disturbances. Concepts such as 'achondroplasia atypica' (SILFVERSKIÖLD), 'chondro-osteo-dystrophy' (BRAILSFORD 1931), and 'dystrophie osseuse familiale' (MORQUIO) may also be included in this general group, although processes of a more penetrative nature affecting the metaphyses are often present and other organic systems may be involved. The individuals affected are often dwarfs and completely disabled.

COCCHI (1950) attempted to assemble some of the penetrative processes under the general term 'polytope enchondrale dysostose'. Changes mainly located in the epiphyses have been given such names as 'dysostosis (enchondralis) epiphysaria' (JANSEN), 'hereditären, multiple Epiphysenstörungen' (RIBBING 1937), 'familiäre multiple Störung der Epiphysenverknöcherung' (MÜLLER 1939), 'dysplasia epiphysealis multiplex' (FAIRBANK), and 'epiphyseal dysplasia' (JACKSON et coll.). Such terms as 'atypical osteochondrodystrophy', or even 'atypical Morquio's disease' are also

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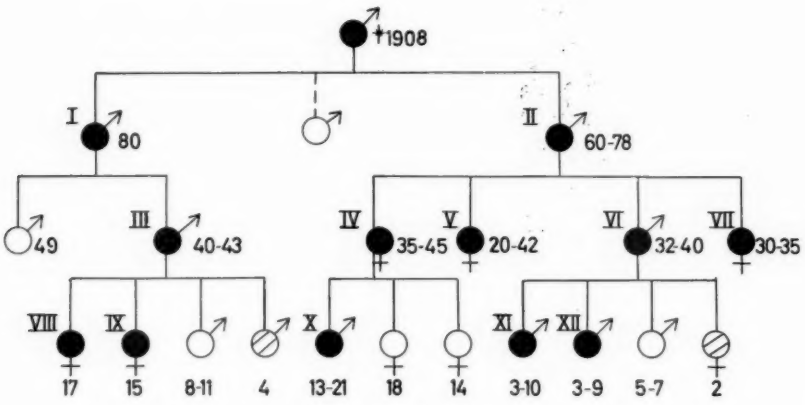

Fig. 1. Genealogic tree.

♀, ♂ = members with hereditary epiphyseal disturbance.

♀, ♂ = uncertain cases (too young to permit a definite diagnosis). Roman numerals denote case numbers in text. Arabic numerals denote ages (in years) between which a person was observed.

encountered. In an endeavour to simplify the terminology, JANSEN, and later WIEDEMANN, suggested the expression 'enchondrale Dysostosen', which has been defined by MAU as 'unkomplizierten, im Charakter nicht tumorartigen, schweren und leichten, metaphysär bis epiphysär gelegenen, also enchondralen konstitutionell-erblichen Knorpelverknöcherungsstörungen des Skeletts'. This concept thus includes endogenous ossification defects of varying degrees, ranging from severe, even fatal cases to benign conditions more in the nature of anomalies.

Roentgenologically, the hereditary epiphyseal disturbances manifest themselves as changes in the ossification pattern of the epiphyses, and the conditions following such changes. The roentgenograms are fairly uniform in character, but certain intra-individual and intra-familial variations have been described. The mode of inheritance also varies. Each case therefore possesses its own particular interest. The present author has made a roentgenologic study of three generations of a family with hereditary, multiple epiphyseal disturbances differing in some respects from those described by other investigators.

Twelve persons were affected. The father of the two oldest in the series was said to have had the same typical symptoms, but he was not examined as he had died in 1908. He had one other son, now dead (middle individual, second generation, Fig. 1), who was healthy. This son's children (4 individuals), grandchildren (10 individuals), and great grand-

a b c

Fig. 2. Case XII in the genealogic tree. a) At 4 years. b) and c) At 9 years.

children (3 individuals) were also well. Several of them have been examined roentgenographically with negative results. Among the affected members of the family there were five not fully grown who were observed for several years together with healthy brothers and sisters and other relations of the same age. The two youngest (Cases XI and XII, Fig. 1) were under observation from the age of 3 to 4 years onwards. The adults in the series (a total of 7 individuals) were also followed over a long period (max. 22 years, Case V).

Roentgenologic findings

Knee joints. The most marked roentgenographic changes, as well as the most prominent clinical signs were situated in the knee joints. In the two youngest subjects (Cases XI and XII), already at 3 to 4 years of age, the distal femoral epiphysis had an irregular porous structure (Fig. 2a), and the bone contour was jagged while that of the border adjoining the metaphysis was smooth. At 9 to 10 years of age, there were several bone centres close to the main nucleus in these cases, the latter being irregular in outline, with protuberances of the type described by Ludloff (Fig. 2b and c). The fragmentation was most marked in the femoral epiphysis but was also present in the proximal nucleus of the tibia, although only in exceptional cases could the changes in this bone be called pathologic. The multipolar ossification of the epiphysis was also striking at 13 years of age; it was particularly noticeable in the

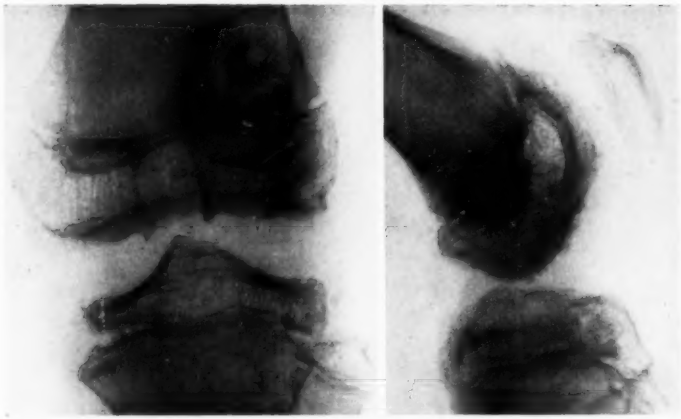

Fig. 3. Case X. At 13 years.

femoral condyles, but the tibial condyles were also affected (Fig. 3). At this age there was also evidence of incipient deformation of the femoral condyles, a condition consistently observed in the adults (Fig. 4a).

Irregularity of the bony structure in the epiphyses was observable during the entire period of growth (Cases X, XI and XII), but it was particularly marked in the prepuberal period, and, in the older members at least, evidence of sclerosis was noted (Fig. 3). In one subject not yet fully grown the bone surface of the patella was uneven (Fig. 3 left). At puberty (Cases VIII and IX) the epiphyses were almost normal in structure. The surfaces of the condyles, however, were still uneven and in many instances isolated bone islands of irregular structure were noted below them, particularly at the 'vertex' of the condyles. The femoral condyles were flattened and the intercondyloid fossa was thus not so well defined as usual in the frontal view; another result of this flattening was malposition of the articular surfaces and irregularity in the height of the 'joint space' (Fig. 5). In the lateral view, the femoral condyles were seen to be roughened and showed fairly typical flattening both in a forward-downward and backward-downward direction (Figs. 4c, 6).

These appearances were also present in the adults. Signs suggestive of osteochondrosis dissecans in various stages were observed and loose bodies were often present. The foci were mostly situated at the vertex of the medial condyle of the femur (Figs. 4a and b, and 5, and 6) but were also seen in the corresponding part of the lateral condyle. In some adults, loose bodies had remained in position for several years and then gradually merged with the surrounding bone (Fig. 4b and c); in others

a b c

Fig. 4. Case X. a) and b) At 18 years. c) At 21 years.

there was practically no roentgenographic evidence of such bodies immediately after the completion of ossification, but they appeared several decades later (Fig. 6). In three adults the patella displayed osteochondrotic changes and an uneven articular surface (Fig. 6). There were irregular, rounded rarefactions in the lateral femoral condyles (Fig. 5) in at least two adults (Cases III and IV). These may have been the remains of osteochondrosis. The condition showed signs of healing. Marked changes of an arthrosis deformans nature had developed in one of the older individuals (Case II) and in at least two subjects the proximal end of the tibia was deformed (Fig. 5).

Ankle joints. The ankle joints came next in importance as regards the extent and severity of the disturbances. At 6 years of age (Cases XI and XII), the outlines of the distal tibial epiphysis and of the trochlear surface of the talus were irregular and this was still more apparent at the age of 9 to 10 (Fig. 7). At the latter age, the trochlear surface also showed upward convexity, or sloped laterally, in the frontal view, and the epiphysis of the tibia showed structural disturbances similar to those in the distal femoral epiphysis, the nucleus displaying irregularities as well as an uneven, jagged outline. The lateral part of the distal tibial epiphysis was not ossified at 9 to 10 years of age, and at 13 years there was fragmentation which only became normal at a comparatively late stage (Fig. 8, left); in several of the adults there was a more or less marked depression at this site (Fig. 9). A few years later (Cases VIII and IX) bone islands were present near the trochlear surface, often at the margins of the trochlea. Osteochondrosis foci were also present in some of the adults, chiefly medially in the trochlea (Fig. 10); the latter was strikingly

Fig. 5. Case IV at 35 years.

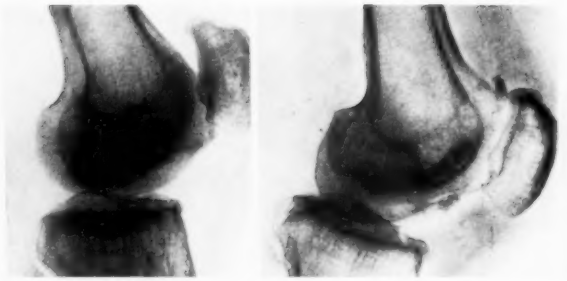

Fig. 6. Case V at 23 years (left) and 42 years (right), respectively.

flat in lateral roentgenograms from the adults (Fig. 10, right). In the a. p. views the trochlea was rounded and the surface sloped laterally in several cases; no degenerative changes were observed in this joint in the adults, but in at least one individual the distal ends of the tibiae were enlarged (Fig. 10, left view).

Feet. In all individuals, both children and adults, the feet displayed the plano-valgus deformity and atypical ossification in the metatarsus. This feature was already present at 6 years of age (Fig. 11, left), perhaps even earlier. The lesions resembled those in the knees and ankles, with irregularity of the bone structure and jagged outlines particularly noticeable at about the age of 9 to 10 (Cases XI and XII, Fig. 11, right). This irregularity of outline was also noted in the bases and heads of the metatarsal bones at an early stage, and at 9 to 10 years of age the changes were striking. At this age also, a pseudo-epiphysis was visible distally in the first metatarsal bone (Fig. 11, right). The trochleae of the phalanges were narrow and roughened in outline in the frontal view, with ossification defects at the margins at an early age (6 years); small isolated bone nuclei or protuberances were present at these sites and the phalangeal epiphyses were in some instances fragmented or irregular at 9 to 10 years of age and some were absent (second phalanx of the fourth and fifth toes (Fig. 11, right)). In a few subjects the epiphyses showed abnormal density (condensation epiphysis). These changes disappeared as ossification progressed. In the fully developed foot, however, a few osteochondrosis foci (Fig. 12) and deformities were still to be seen. In the adults, loose bodies were observed mostly at the surface of the head of the talus (Fig. 10, left) and in the first, second and fifth metatarsal heads (Figs. 12, 13). The deformed metatarsal heads were often flattened and their surfaces irregular, and at the zone bordering on the diaphyses there were clearly delineated ridges (Fig. 13). In a few individuals the bases of the phalanges

Fig. 7. Case XII at 9 years.

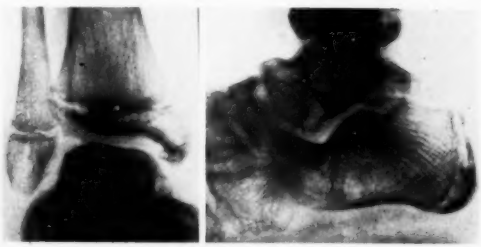

Fig. 8. Case X at 13 years.

were also enlarged and the surfaces irregular (Fig. 13). Accessory bones were often seen, and in one subject (Case VI) there was a persistent apophysis at the tuberosity of the fifth metatarsal.

Elbow joints. The surface of the capitulum humeri was irregular and roughened already at 6 years of age (Cases XI and XII), the other joint surfaces being less affected. At a later stage in the bone development the surfaces were seen to be extremely uneven, almost 'fringed', and the epiphyseal nuclei, especially of the trochlea and capitulum humeri, irregular in structure (Fig. 14). At the age of 9 to 10 there were also a number of bone islands near the different epiphyses, as well as ridgelike protuberances close to the main centre. These disturbances were in all individuals most noticeable in the trochlea humeri and the olecranon. The head of the radius, on the other hand, could with certainty be said to be the seat of such changes only in one child (Fig. 15). The trochlear epiphysis was ossified within the normal time on the ulnar side only, while the radial part was delayed; it developed multilocularly, and was still fragmented at 13 years of age (Fig. 15, left). This area seldom reached normal development even in the adults (Fig. 16, cf. p. 108). The bone structure in the epiphyses was irregular and sclerotic in the prepuberal years (Fig. 15). The epicondylar epiphyses were also irregular, and that on the ulnar side was distally situated close to the nucleus of the trochlea (Fig. 14). When the epiphyseal lines were nearly or completely united, at 15 to 17 years of age, some irregularity was still observable in the olecranon, but the surfaces were now smoothly outlined and the bone structure nearly normal. In the adults, only slight traces remained to tell of disturbed epiphyseal development. An important observation, however, was that in every instance the bone surface of the humerus was roughened and there was mal-articulation with the ulna and radius (Fig. 16). This was partly due to the fact that the surface of the head of the radius sloped towards the ulna. In two individuals the head of the radius was asymmetric with large ridges distally; two subjects had loose bodies in the joint without sign of corresponding defects in the bone.

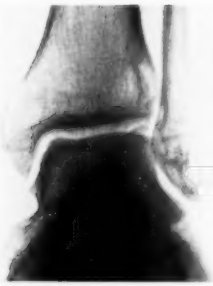

Fig. 9. Case VIII at 17 years.

Fig. 10. Case III at 40 years. Osteochondrosis of talus (arrow).

Wrists and hands. There was disturbance of the ossification in the distal radial and ulnar epiphyses in a few of the subjects (Fig. 17). At the age of 6 years the lesions in the wrists and hands were similar to those in the foot. The heads of the metacarpals were uneven, and pseudo-epiphyses were distinguishable distally in the first metacarpal bone. The trochlear margins of the phalanges were rounded and imperfectly ossified (Fig. 18). The most marked roentgenologic changes were observed at the age of 9 to 10 (Fig. 19). The carpal bones were also involved and among other features, the scaphoid showed fragmentary ossification (Fig. 17). The metacarpals were jagged in outline and irregular in structure at both the proximal and distal ends, and there were accessory bone nuclei either lying entirely free or forming protuberances. The phalangeal epiphyses were also fragmented or atypical in shape and structure in some individuals. There was, for example, a press-button-like variant (Fig. 19, distal phalanges of second and third finger). The appearances were similar at a later stage, although ossification had now progressed; enlarged metacarpal bases and heads were still observed. In addition, the bony structure was sclerotic, with hypertrophic bone trabeculae, especially in the carpus (Fig. 17).

The situation had changed in the adults. With few exceptions (Fig. 21) the distal radial and ulnar ends were normal. The heads of the metacarpals (especially the first and second) were, on the other hand, deformed and irregular, and their surfaces were slightly uneven and flattened: in only one case, however, was there a definite osteochondrotic focus (Fig. 20, second metacarpal). The metacarpal heads, like those of the metatarsals, were distinctly delineated at the diaphyses owing to the presence of unusually prominent marginal ridges (Fig. 21).

The other joints were roentgenologically normal in all individuals. No defects or deformities were observed in other organs.

Fig. 11. Case XII at 6 years (left) and 9 years (right), respectively.

Fig. 12. Case VIII at 17 years.

Fig. 13. Case III at 40 years.

Clinical aspects

The roentgenologic lesions were reflected in typical clinical manifestations. These appeared first around the ages of 3 to 4 years in the form of walking difficulties and gradually became aggravated until, between 7 and 14 years of age, when the roentgenologic changes were most marked, much discomfort, particularly in the knee and ankle joints, was experienced in connection with locomotion. The gait was staggering and unbalanced, and transference of weight was not borne correctly by each foot. Certain weight-bearing movements of the knee joints such as 'knees bend' were executed with great difficulty. Another typical feature was that the knee and ankle joints became stiff after sitting; this was more severe if muscular effort had preceded the rest period (cf. RIBBING 1937).

These disabilities decreased during puberty but they were still present in some degree in the adults. The clinical signs were proportional to the roentgen appearances. Young individuals with marked epiphyseal changes during growth were more or less disabled up to the time when the epiphyses had become completely ossified. When this stage was reached, a clinical improvement was noted. The adults presented signs of osteochondrosis dissecans (locking of the joints, transient extension difficulties) but also suffered from flatfoot. The knee joints in particular seem to have been affected; three subjects, for example, had been operated on for loose bodies in this joint (Figs. 5, 6). The surgical records described typical features of osteochondrosis. Deformity of the articular surfaces, suggesting an adjustment of shape to meet irregularities in underlying

Fig. 14. Case XI at 10 years.

Fig. 15. Case X at 13 years.

Fig. 16. Case VI, at 40 years.

bones, was not observed. In other joints, for example the ankle joints and metatarso-phalangeal articulations (Figs. 12, 13), locking and limitation of movement were reported by the adults. Three had signs of hallux rigidus (cf. RIBBING 1937). In the elbow joints of two individuals, large loose bodies periodically caused considerably limited movement. In general, however, the joints of the upper extremities do not seem to have produced symptoms either in children or adults; even joints with marked roentgenologic changes caused no particular trouble.

Clinical examination revealed little to suggest these skeletal changes. The children's joints were completely normal except for slight crepitations. The latter sign was fairly marked in some of the adults, especially in the knee joints. When osteochondrosis with loose bodies was present these could often be palpated.

Discussion

The disease picture was uniform in all these individuals. No abortive forms were encountered, nor were there any bone changes in healthy relations detectable by roentgenography alone. Such changes are, however, sometimes encountered in enchondral dysostosis (RIBBING 1937, HERMODSSON, BRAILSFORD 1953, MAU, and others), especially in the more penetrative types. In these, healthy relations of affected persons may display a remarkable incidence of pseudo-epiphysis, considerable epiphyseal fragmentation during the ossifying period, and often numerous accessory bones.

In the family discussed here, the lesions in the epiphyses of the different joints showed strong resemblances and for the most part only small individual variations were observed. A few characteristic features such as a strong tendency to osteochondrosis in certain joints, and deformed metaphyses, were probably ascribable to such features as mil[illegible]u

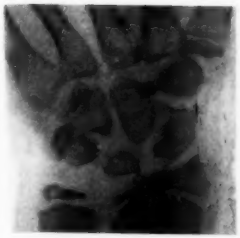

Fig. 17.

Fig. 18.

Fig. 19.

Fig. 17. Case X at 13 years. Scaphoid fragmented proximally.

Fig. 18. Case XII at 6 years.

Fig. 19. Case XI at 10 years.

or sex. The bilateral nature of the changes was also noteworthy in the material. Homologous joints such as the elbow and knee joints and the distal joints of the metatarsals and metacarpals were largely involved. The disturbances in the surfaces at the lower ends of the humeri and femurs had several features in common (Figs. 5, 16).

RIBBING and MÜLLER 1933 and 1939, and other investigators, have made similar observations. A symmetric location would also seem to be a characteristic of hereditary epiphyseal disturbances. In more penetrative bone conditions involving the metaphysis, symmetry is not so noticeable a feature, possibly because secondary factors such as uneven weight-bearing conditions play a more prominent part.

The extent to which the metaphyses were involved in my cases is not quite clear. It is true that the ends of the bones were enlarged around the deformed joints and that in at least four adults the ends of certain bones such as the metatarsal and metacarpal heads were deformed (Figs. 5, 10, left, 13, 21), but these observations need not necessarily indicate a primary metaphyseal disturbance; they might be due to an abnormal, formative influence of the epiphyses, or to insufficiency reactions. The same is probably true of the sporadic changes noted in the diaphyses. The severe flatfoot is the only sign suggesting a deeper involvement of the metaphysis; although flatfoot is usually regarded as a primary weakness of the ligaments and soft tissues and is often unassociated with enchondral dysostosis, the combination is a common one. Other conditions of approximately the same type, such as genu valgum,

Fig. 20.

Fig. 20. Case V at 40 years. Osteochondrosis of head of second metacarpal.

Fig. 21. Case III at 41 years.

Fig. 21.

coxa vara, and kyphosis, may likewise occur in conjunction with hereditary epiphyseal disturbances (MAU). Thus it is probably not possible to make a clear distinction between primary metaphyseal and epiphyseal disturbances; presumably, it is a question of an imperceptible transition, as MAU, MARQUARDT, and other workers, have already pointed out.

The roentgenograms were characterized by changes in the epiphyseal structure. This was coarse at the beginning of the ossifying period (Figs. 2, 7, 14), and at a later stage (in prepuberty) there were definite signs of sclerosis (Figs. 3, 8, 15, 17) which were probably attributable to functional hypertrophy of the bone trabeculae (cf. WOLFF's law of transformation).

The original centres of ossification tended to be atypical in shape and in a few cases in location as well. A multilocular disposition, often in the form of one main centre with small accessory bone islands eccentrically situated, was also a characteristic observation in my cases. MAU has pointed out that this is the typical finding in those forms of enchondral dysostosis in which the epiphysis is mainly involved. Local delay in the ossification (left views of Figs. 7, 8, 10, and 15, and Figs. 14 and 16)

was observed in certain parts of the nucleus, typical for all individuals, but on the whole even the most fragmented epiphyses became ossified within the normal time. Nor was the closure of the epiphyseal lines delayed. This observation is also in agreement with earlier descriptions of hereditary multiple epiphyseal disturbances (RIBBING 1937, MÜLLER 1939, MAU, and others).

In many of the adults in the present series, the bone surfaces of the joints were characteristically deformed in relation to the opposing surfaces. The individual likenesses in this respect were often remarkable, even between relatively distant relations (left views of Figs. 8 and 10). RIBBING 1933, 1937, FAIRBANK, MÜLLER 1939, and other investigators have maintained that an increased distance between the bone surfaces is a typical feature of epiphyseal disturbances. A perusal of the literature reveals, however, that deformation of the epiphyses varies in different families and also between different members of one family. This may have something to do with the advanced nature of the enchondral dysostosis process.

There were certain roentgenologic features in my material which, if taken alone, could hardly be described as pathologic but rather as clinically irrelevant anomalies or physiologic variations. Among these may be mentioned pseudo-epiphyses, condensation of the phalangeal epiphyses, and accessory bones; multiple centres of ossification may also be included here (Figs. 11, 17, 19) (cf. LIESS, MAU, and others). These abnormalities were, however, both marked and numerous, simulating real disease. Similar points of view have been expressed in earlier reports on hereditary epiphyseal lesions (RIBBING 1937, MÜLLER 1939, and others). It has also been assumed that constitutional epiphyseal weakness (LEHMANN) might be a common etiologic factor in producing enchondral dysostosis and the variants mentioned. In other words, the difference between these conditions would be only quantitative. The hereditary epiphyseal disturbances may be regarded as an excessive exaggeration of what is otherwise called ordinary or 'normal' development of bone.

In most of the publications on hereditary disturbances of epiphyseal development the hip and shoulder joints and the vertebrae are the sites said to be most frequently involved, as well as those showing the most advanced changes. It is in these areas also that the most disabling features have been observed (RIBBING, MÜLLER, ROMANUS, HERMODSSON, FAIRBANK, WATT, CLÉMENT, MAUDSLEY, JACKSON et coll., WAUGH, LEVY et coll., CHRISTENSEN et coll., FREIBERGER, MAU, and others). My cases were unusual in that only peripheral joints of the extremities were involved. Another characteristic was that the clinical symptoms and signs were also located in the joints of the lower extremities and, furthermore, these signs appeared earlier than in the cases described by others, *e. g.*

RIBBING 1937. There are, however, a few endogenous epiphyseal disturbances of a similar nature in which the changes are located in the phalanges (THIEMANN's disease) or in the metatarsals and metacarpals (DIETRICH's disease); in other words, an even more peripheral site than in my material. These conditions are relatively benign and without metaphyseal disturbances, although concomitant changes in the femoral head and the vertebrae may occur (COCCHI 1952, MEISENHEIMER). THIEMANN's and DIETRICH's diseases are often dominantly inherited (COCCHI 1952), as was the case with my material. On the other hand, it has been stated that the 'central' hereditary multiple epiphyseal disturbances are recessively inherited (RIBBING 1937, MÜLLER 1939, JACKSON, FAIRBANK, MAU, LEVY et coll., and others). Probably, my cases represented an intermediary stage between the relatively serious disturbances mainly localized to the epiphyses of centrally situated bones, and the milder forms of the THIEMANN and DIETRICH type. They displayed features pointing in both directions, but they possessed certain characteristics both as regards the site of the lesions and their nature. For instance, fairly marked changes were present in the knee joints in particular, as well as in the talocrural joints and the joints of the feet during the period of ossification, giving rise to local symptoms. In the adults, the roentgenologic and clinical findings were dominated by osteochondrosis, especially in the knees and other weight-bearing joints. This condition was present less frequently in the joints of the upper extremities (cf. the metatarsal and metacarpal joints), an observation suggesting that the healing tendency was greater and the influence of external factors less marked in these joints. These osteochondroses recall certain descriptions — especially by early investigators — of multiple, possibly familial osteochondroses (WRIGHT, STÖREN, MÜLLER 1933, MÜLLER & HETZAR), which RIBBING 1937 and MÜLLER at a later date (1939), endeavoured to classify as hereditary epiphyseal disturbances. In the opinion of the last two authors the only difference between these conditions was one of degree. Hereditary multiple aseptic necrosis has also been discussed in connection with the same group of diseases by RIBBING, MÜLLER (1939), HERMODSSON, CLÉMENT, MAU, and other investigators. From findings in cases of varying severity these authors considered that these conditions have a common denominator, namely, LEHMANN's constitutional epiphyseal weakness. My cases would seem to illustrate this connection more clearly than those of earlier workers, especially as regards the subjects with osteochondrosis. The foci were often situated at the sites of predilection, and nothing in the clinical findings diverged from genuine osteochondrosis (cf. RIBBING 1937, 1955). If we accept LEHMANN's theory, then these cases may possibly by regarded as an intermediary stage between the centrally situated hereditary multiple epiphyseal

disturbances with deformity in the vertebral column, hip joints and shoulder joints, and the multiple — in some cases hereditary — osteochondroses described by WRIGHT, MÜLLER (1933), MÜLLER & HETZAR, NOVOTNY, LÖTHMAN & NORDSTRÖM, GARDINER, PICK, and others. The present paper demonstrated the value of studying epiphyseal disturbances of a mild nature by repeated examinations covering both the ossification period and the subsequent osteochondrosis 'age'.

SUMMARY

Twelve members of a family with hereditary epiphyseal disturbances have been studied by roentgenography. The observations accorded with the descriptions of other investigators. Contrary to earlier reports, the changes were situated only in the joints of the extremities and the clinical picture in the adults was dominated by osteochondrosis.

ZUSAMMENFASSUNG

Zwölf Mitglieder einer Familie mit heriditären Epiphysenstörungen sind röntgenologisch untersucht worden. Die Beobachtungen standen mit der Beschreibung anderer Untersucher im Einklang. Im Gegensatz zu früheren Berichten waren die Veränderungen nur in den Gelenken der Extremitäten gelegen und das klinische Bild bei Erwachsenen wurde von der Osteochondrose dominiert.

RÉSUMÉ

Douze membres d'une famille atteinte d'anomalies épiphysaires héréditaires ont été examinés radiographiquement. Les signes observés sont conformes à la description d'autres auteurs. Contrairement à des publications antérieures, les lésions siègent uniquement sur les articulations des extrémités, et chez les adultes, le tableau clinique est dominé par l'ostéochondrose.

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ANGIOGRAPHIC DEMONSTRATION OF THE POSTERIOR COMMUNICATING AND POSTERIOR CEREBRAL ARTERIES

II. Pathologic angiography

by

Georg-Fredrik Saltzman

The demonstration of the posterior cerebral artery by carotid angiography in different pathologic states has been discussed by a few investigators. CHRÁST & VÁGNER considered that this vessel could be more frequently demonstrated in cases of brain tumour than in normal. ENGESET also obtained supporting evidence for this view. FERNANDES & ALVES noted an increased incidence of filling of the posterior cerebral artery in a series of cases of oligophrenia. They suggested arrested development as an explanation, drawing attention to observations showing that the posterior communicating artery is wider in the foetus and in newborn infants than in adults (DE VRIESE).

Certain forms of intracranial pathologic states were represented in such large numbers in our angiographic material that there seemed to be justification for investigating the incidence of demonstration of the

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posterior communicating and posterior cerebral arteries. The material treated in this paper consists of 223 angiographies in patients with intracranial expansive lesions, 118 examinations in arterial aneurysm, 36 in arteriovenous aneurysm, and 21 in thrombosis of the internal carotid artery system situated distal to the origin of the posterior communicating artery. No subdural haematomas were included among the intracranial expansive lesions. Only conventional carotid angiographies were studied in this investigation.

For comparison with a 'normal' material and information on the principles underlying the statistical analysis of the material, the reader is referred to part I.

Intracranial expansive lesions

General observations

In 223 carotid angiographies carried out on the side on which an expansive process was situated or was most advanced the posterior cerebral artery was visible in 78 instances (35.0 ± 3.2 %). This incidence is almost in complete accord with the corresponding figure in the normal material.

When the material was divided into internal carotid and common carotid angiographies, slightly different findings were obtained. There were 101 internal and 122 common carotid angiographies in the material. The posterior cerebral artery was visible in 46 (45.5 ± 5.0 %) of the internal carotid and in 32 (26.2 ± 4.0 %) of the common carotid examinations. In this material also, the difference between the internal and common carotid angiographies is significant.

The posterior communicating artery was demonstrated in 13 instances (5.8 ± 1.6 %) without the posterior cerebral artery being simultaneously visible. The incidence was the same in both the internal and common carotid angiographies, and was as high as in the normal material.

Retrograde filling of the basilar artery was noted in 5 instances (2.2 ± 1.0 %). Of these, 4 were internal carotid and one a common carotid angiography. The incidence was slightly higher than in the normal material but the difference is not significant.

Contrast medium was injected into both carotid arteries in 26 patients with intracranial expansive processes. The posterior cerebral artery was filled on the side on which the lesion was situated in 14 cases and on the opposite side in 12. The difference is not significant.

Table 1

Demonstration of the posterior cerebral artery in intracranial expansive processes at different sites

Site of expansive lesion	No. of cases	Filling of posterior cerebral artery	
		No.	%
Frontal	96	38	39.6 ± 5.0
Parietal	43	16	37.2 ± 7.4
Temporal	45	9	20.0 ± 6.0
Central	22	10	45.5 ± 10.6
'Others'	17	5	29.4 ± 11.1
Total of cases and mean percentage	223	78	35.0 ± 3.2

Site of the expansive lesions

Because of the small size of the material only a rough classification was made in respect to the site of the lesions. A more detailed classification would have resulted in a large number of groups with so few cases in each that a statistical analysis would have been impossible. The expansive lesions were divided into frontal, parietal, temporal, and central processes. Lesions encroaching upon two of these regions were placed in the group corresponding to the site where the lesion was most extensive. Another group headed 'Others' contained miscellaneous occipital, cerebellar, and multiple lesions, as well as processes involving more than two of the afore-mentioned regions. The distribution of the cases as well as the extent to which the posterior cerebral artery was visible in the different groups will be seen from Table 1.

A comparison of the incidence of a contrast-filled posterior cerebral artery in the different groups with the corresponding figure for the whole series of expansive lesions revealed no significant difference between the frontal and parietal processes and the total material. The difference was greater for the central processes but not significant here either. It may be mentioned that FISCHER also noted that the posterior cerebral artery was more frequently seen in cases of tumour with a central site. In patients with temporal processes the posterior cerebral artery was less often demonstrated than in any other group. This latter observation is in direct contrast to CHRÁST & VÁGNER's assertion that the posterior cerebral artery is more often demonstrated in temporal tumours than in tumours in any other situation. It is true that expansive lesions of types other than tumours are included in our material, but even when only the tumours were considered, the posterior cerebral artery was less frequently visible in the temporal group in our material than at any other site. Not even here, however, is the difference significant. This material of intracranial expansive lesions is not large enough to permit differences of this magnitude to be statistically established.

Table 2

Demonstration of the posterior cerebral artery in different intracranial expansive lesions

Nature of expansive lesion	No. of cases	Filling of posterior cerebral artery	
		No.	%
Glioma	96	34	35.4 ± 4.9
Meningioma	41	16	39.0 ± 7.6
Intracerebral haemorrhage	26	5	19.2 ± 7.7
'Others'	32	9	28.1 ± 7.9
Expansive lesions of undiagnosed type	28	14	50.0 ± 9.4
Total of cases and mean percentage	223	78	35.0 ± 3.2

Nature of the expansive lesions

In the classification of the expansive lesions according to their nature the number of groups was also kept as small as possible in order that a statistical analysis could be made. The gliomas, which constitute the largest group, were therefore not divided up into their histologic subgroups but were treated as a whole. Other groups large enough to be evaluated separately were the meningiomas and intracerebral haemorrhages. In some cases the nature of the lesion had not been diagnosed with certainty. All other diagnoses had been verified at operation or autopsy, and most of them histologically as well. A few uncommon forms of expansive lesions such as metastases, abscesses, cysts, angiomas, sarcomas, cholesteatomas, and tuberculomas were placed in a group headed 'Others'. The distribution of the cases in the various groups as well as the incidence of demonstration of the posterior cerebral artery will be seen from Table 2.

The posterior cerebral artery was visible in the glioma group to the same extent as in the whole material of expansive lesions. The incidence was slightly higher for meningiomas but the difference is not significant. In intracerebral haemorrhage, the posterior cerebral artery was less often seen than in other expansive changes. The difference is not significant, however. It is worthy of note that the posterior cerebral artery was not visible in a single one of the 7 cases of temporal haemorrhage. The artery was more often demonstrated in the group containing undiagnosed expansive lesions than in the rest of the material. The difference is almost significant, but owing to the nature of the group the observation could not be used as a basis for conclusions. Hence, here too, the groups are not large enough to permit small differences to be statistically established.

Paying consideration only to the part of the material consisting of true verified tumours, *i. e.* gliomas, meningiomas, metastases, and sarcomas, it was noted that the posterior cerebral artery was visible in 55 out

of 149 cases (36.9 ± 4.2 %). There is no significant difference either in relation to the whole material of intracranial expansive lesions or to the normal material.

Conclusions. Contrary to the observations of other authors, our material furnished nothing to indicate that the posterior cerebral artery is generally more often visible in tumour cases than in normal subjects. The same may be said of intracranial expansive lesions on the whole. The centrally situated lesions showing a slightly higher incidence, although not a significant difference, form, perhaps, an exception to this rule, but no plausible explanation can be given.

A lower incidence of visible filling of the posterior cerebral artery was noted in temporal lesions than in either the normal material or the whole material of intracranial expansive processes. A compression effect on the posterior communicating artery and/or the posterior cerebral artery may constitute a conceivable explanation of this observation. The difference is, however, not statistically established.

The posterior cerebral artery was also relatively seldom demonstrated in cases of intracerebral haemorrhage. It is tempting to discuss the possibility of a connection between this feature and intracranial spasm. ECKER & RIEMENSCHNEIDER observed a very high incidence of spasm in non-traumatic intracerebral haemorrhage.

Arterial aneurysm

General observations

The posterior cerebral artery was filled in 32 (27.1 ± 4.1 %) of the 118 carotid angiographies in which arterial aneurysm was demonstrated. The incidence was lower than in the normal material, but the difference is not significant. Retrograde filling of the basilar artery was not observed at all.

Of these examinations, 62 were internal carotid and 56 common carotid angiographies. The posterior cerebral artery was filled in 19 (30.6 ± 5.9 %) of the former and in 13 (23.2 ± 5.6 %) of the latter. Both of these incidence figures were lower than the corresponding figures in the normal material. The difference is greater for the internal than for the common carotid angiographies.

Extravascular injection of contrast medium was noted in only 4 instances, and this had no effect at all on the incidence of filling of the posterior cerebral artery. The incidence of haematoma in connection with the puncturing could not be ascertained.

Table 3

Demonstration of the posterior cerebral artery in arterial aneurysm at different sites

Site of aneurysm	No. of cases	Filling of posterior cerebral artery	
		No.	%
A origin of posterior communicating artery	33	15	45.5 ± 8.7
A bifurcation of interior carotid artery	7	—	
A origin of anterior communicating artery in anterior cerebral artery	40	9	22.5 ± 6.6
I middle cerebral artery	32	6	18.8 ± 6.9
I pericallosal artery	4	2	
I extradural part of interior carotid artery	2	—	
Total of aneurysm cases and mean percentage	118	32	27.1 ± 4.1

In 4 instances (3.4 ± 1.7 %) the posterior communicating artery was visible, but not the posterior cerebral artery. The incidence for this finding also was thus lower than in the normal material.

Grouping the cases with filling of the posterior cerebral artery according to the relation between the widths of the posterior communicating and posterior cerebral arteries, the same distribution over the three groups was noted as in the normal material (cf. part I). The figures are too small to justify a statistical analysis.

Site of the aneurysms

Aneurysms are usually situated close to vascular bifurcations. The angiographically demonstrable arterial aneurysms could usually be placed according to their site in one of the following four groups:

1. Aneurysm of the internal carotid artery at the origin of the posterior communicating artery.

2. Aneurysm at the site where the internal carotid artery divides into the anterior cerebral and middle cerebral arteries.

3. Aneurysm at the site where the anterior communicating artery leaves the anterior cerebral artery.

4. Aneurysm of the middle cerebral artery.

In addition to aneurysm at these sites there were also 4 cases in which the aneurysm was situated in the pericallosal artery at the origin of the callosomarginal artery and 2 in which it had originated in the extradural, intracranial part of the internal carotid artery. Other sites are also encountered but they are uncommon and not represented in this material. The distribution of the aneurysms over these four groups and information

on the filling of the posterior cerebral artery in the different groups will be seen from Table 3.

In the three groups large enough to warrant a statistical analysis, the posterior cerebral artery was visible more often than normally in those cases in which there was aneurysm at the origin of the posterior communicating artery. When the aneurysm was in some other position, however, the posterior cerebral artery was not so often shown as in the normal material. The difference between aneurysm at the site where the posterior communicating artery leaves the internal carotid artery and other aneurysms is significant. If these aneurysms at the origin of the posterior communicating artery are deducted, the others show such a low incidence for filling of the posterior cerebral artery that the difference as against the normal material is significant.

Conclusions. The posterior cerebral artery is generally demonstrated less frequently in an aneurysm material than in a normal series. It seems plausible to suspect that this observation may have some connection with the vascular spasm noted by ECKER & RIEMENSCHNEIDER and LÖFSTEDT in a large percentage of arterial aneurysm cases.

Cases with aneurysm of the internal carotid artery at the origin of the posterior communicating artery form an exception to this rule. The explanation of this is probably anatomic. It is conceivable that the close proximity of the aneurysm may in some way hinder the posterior communicating artery from contracting. It is not unlikely that the muscle layer of the vessel wall might be defective in the posterior communicating artery, at least in the segment closest to the internal carotid artery, in other words, in the immediate vicinity of the aneurysm. Such lesions have been described by HASSLER & SALTZMAN in a preliminary report on widening of the infundibular part of the posterior communicating artery.

Arteriovenous aneurysm

It is obvious that a malformation swallowing such large amounts of blood as an arteriovenous aneurysm in the vascular region served by the posterior cerebral artery must receive blood from both vertebral and carotid arteries. Visible contrast-filling of the posterior cerebral artery is always obtained in these cases at carotid angiography. The widest posterior communicating arteries observed at carotid angiography were encountered among the 6 cases of this type in the material.

Among the other 30 cases the posterior cerebral artery was demonstrated in 10 (33.3 ± 8.6 %). The incidence was almost the same as in the normal material. The material does not permit a more detailed analysis.

Thrombosis in the internal carotid artery system

The material consists of 22 cases of thrombosis in the internal carotid rtery system distal to the origin of the posterior communicating artery. he posterior cerebral artery was demonstrated in 14 instances (63.3 ± 0.3 %). The incidence of filling of this vessel was thus much higher han in the normal material. The difference is significant.

The thrombus was located in the upper part of the carotid siphon in instances, in the anterior cerebral artery in one case, and in the middle erebral artery in the other 18. The posterior cerebral artery was filled i all three patients with a thrombus in the carotid siphon and also in the patient with thrombosis of the anterior cerebral artery.

The posterior communicating artery was narrower than the posterior erebral artery in 10 of the 14 cases in which the latter vessel was demonstrated. In 4 of the cases, the diameter of the posterior communicating artery was only half that of the posterior cerebral artery, or even narrower. Thus, in these thrombosis cases, a considerable number of vessels were filled which, as shown in part I, because of the relatively small width of the posterior communicating artery are comparatively seldom shown under normal circumstances in 'conventional' carotid angiographies.

SUMMARY

The angiographic demonstration of the posterior cerebral artery has been studied in intracranial expansive lesions, consideration having been paid to the nature and site of the processes. The vessel was similarly studied in arterial aneurysms and in a small series of cases of arteriovenous aneurysm and of thrombosis in the internal carotid artery system distal to the origin of the posterior communicating artery. The incidence figures are compared with the corresponding figures in the normal material.

ZUSAMMENFASSUNG

Die angiographische Darstellung der Arteria cerebralis posterior ist bei intrakraniellen expansiven Erkrankungen untersucht worden, wobei die Natur und die Lage des Prozesses beachtet wurde. Das Gefäss wurde gleichartig bei arteriellen Aneurysmen, bei einer kleinen Anzahl von arteriovenösen Aneurysmen und bei Thrombose im Ausbreitungsgebiet der Arteria carotis interna, distal vom Abgang der Arteria posterior communicans, untersucht. Die Ergebnisse werden mit entsprechenden Erfahrungen an einem Normalmaterial verglichen.

RÉSUMÉ

L'auteur étudie la mise en évidence angiographique de l'artère cérébrale postérieur dans les lésions expansives intracraniennes, en tenant compte de la nature et du siège d ces affections. Il a étudié de la même façon ce vaisseau dans les anévrismes artériels e dans de petites séries de cas d'anévrismes artério-veineux et de thromboses du système carotidien interne situées en aval de l'origine de l'artère communicante postérieure. I compare la fréquence de la mise en évidence de cette artère dans ces affections avec l fréquence dans les cas normaux.

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ROENTGENOLOGIC FINDINGS IN ULCERATIVE COLITIS WITH MALIGNANT DEGENERATION

by

N. P. G. Edling, R. Lagercrantz and *H. Rosenqvist*

Clinical statistics indicate that malignant degeneration occurs in about 3 to 4 % of cases of ulcerative colitis (SVARTZ & ERNBERG 1949, LAGERCRANTZ (only children) 1955, KIRSNER 1957, SVARTZ & GILLNÄS 1958). The incidence of this change, however, increases with the duration of the disease, so that in patients with a history of ulcerative colitis for more than 10 years, the figure becomes 37 % (BACON 1948, LYONS & GARLOCK 1951, COUNSELL & DUKES 1952, CATTELL & COLCOCK 1955). In a series of 150 children LAGERCRANTZ 1955 found no signs of malignant degeneration in 77 cases examined 10 years after the onset of the disease, but out of 24 cases with a duration of 15 years 8 were found to have developed malignant changes (33.3 %).

Armed with these facts, it was considered to be of interest to determine whether or not there were any typical roentgenologic features in a series of patients with ulcerative colitis who subsequently developed malignant degeneration. The present authors have reviewed the clinical aspects of 26 such cases in another paper, and the roentgenologic investigations which were then not discussed at any length are now presented.

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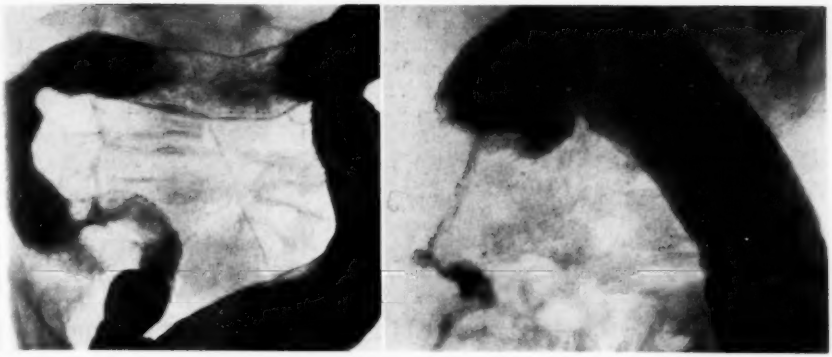
Fig. 1. Ulcerative colitis + carcinoma. Reduced distensibility and absence of haustrations. Irregular cancerous stricture in distal part of transverse colon.

In 14 of the cases the entire colon showed signs of severe chronic inflammatory changes when the diagnosis of carcinoma was made (Fig. 1). The colon was short, in most cases without loops, and in a few cases with loops which were much shorter than normal. The size of lumen was reduced, and there was a high degree of dehaustration. The contours of the bowel were usually smooth, and where the thickness of the colon wall could be estimated it was found to be greater than normal. There was only one case of polyposis, and this only to a slight degree; at autopsy there was no evidence of malignant degeneration of the areas involved. In no case was there any evidence of pseudopolyposis. In 7 cases, examined twice or more before a growth developed, no improvement was demonstrable other than a change from an acute or subacute to a chronic stage of inflammation.

In 4 further cases similar signs of ulcerative colitis in the entire colon were found at roentgen examinations three to six years before a carcinoma was discovered at operation or autopsy; at autopsy three of the cases still showed colitic changes. The fourth case was explored but no autopsy performed.

The remaining 8 cases form a more interesting and important group. At the first roentgen examination 5 of these had involvement of the entire colon similar to that described above, and 1 had such changes in the distal part of the colon (Figs. 2 and 3). On subsequent examinations the length of the colon was increased and the loops were again present, the appearances at times being normal. The distensibility also appeared to be improved and haustrations were visible. The roentgen findings indicated that a true improvement or healing of the inflammation had oc-



a b

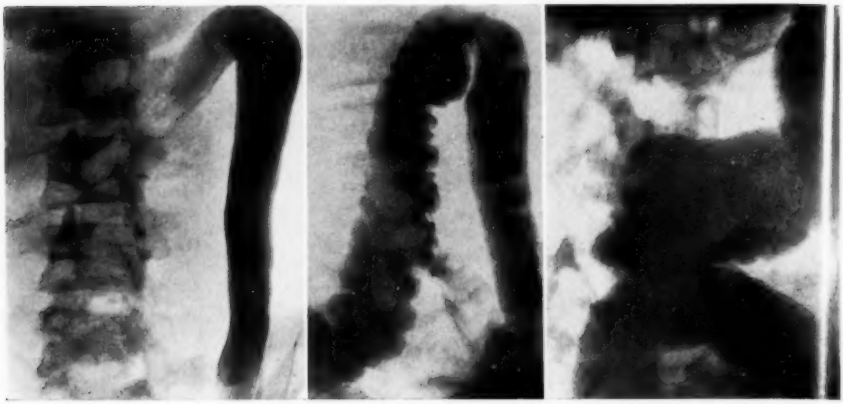
FIG. 2. Ulcerative colitis + carcinoma. a) 1941: Short colon, reduced distensibility, absence of haustrations, and irregular contours. b) 1957: Colon longer and distension improved. Irregular cancerous stricture in distal part of transverse colon.

curred even as the malignant degeneration appeared. The case histories of the remaining 2 patients pointed towards ulcerative colitis for several years, but the roentgen findings were normal except for the cancerous strictures.

In 23 of the cases, the inflammatory changes involved or had at some time involved the entire colon. In one case only the distal part of the colon was affected. No final assessment was possible in the remaining 2 cases.

As to the age when the carcinoma was discovered in the first 18 patients, 13 were between 17 and 31 years of age and 5 between 45 and 58 years. In this series 6 patients were females, three of them belonging to the younger group and three to the older group. In the second group of 8 patients 6 were between 22 and 29 years of age and 2 between 40 and 46 years. Three of the patients in the younger group and both the patients in the older group were females.

It is noteworthy that the material may be divided into two age groups: 19 patients between 17 and 31 years of age, and 7 patients between 40 and 58 years of age. In the entire material the males preponderate, whereas in the older group most of the patients are females. The younger patients in both groups had the onset of their ulcerative colitis in childhood or in early youth. In the older group (40 to 58 years) the duration from the onset of the ulcerative colitis to the advent of malignant degeneration was more than 5 and up to 30 years, with an average of about 14 years. The average duration of the disease until the discovery



a b c

Fig. 3. Ulcerative colitis + carcinoma. a) 1941: Colon shorter and less distensible than normal. Absence of haustration. b) 1949: The bowel is longer and better distended. Now, only sigmoid and descendens are dehaustrated. c) 1958: Length, distension, and haustrations normal but irregular cancerous stricture in mid transverse colon.

of malignant degeneration for all patients was 15.4 years. More than three-quarters of the patients had very slight or no symptoms referable to the colitis when those of the malignant complication appeared.

The macroscopic appearance of the carcinomas in the present material has been obtained from operative reports, from pathologico-anatomic studies of specimens obtained at operation or autopsy, and/or from the roentgenologic findings. In one case there were two tumours, one in each flexure. In all the cases the tumours were of a common type, well-demarcated from the adjacent non-malignant bowel. Thus, when compared with the usual carcinoma of the colon, there is no characteristic roentgen appearance of malignant change in ulcerative colitis in the present material.

Microscopically, most of the tumours have been highly or moderately differentiated adenocarcinoma. A few have been poorly differentiated and none had been scirrhous. Eight of the tumours were colloid-secreting carcinomas.

In this series the localization of the tumours does not follow the usual distribution of carcinoma of the colon published in text-books. Only 4 (15 %) of the 27 tumours were situated at the recto-sigmoid junction, and 23 (85 %) in other parts of the colon, usually in the transverse colon or one of the flexures. This distribution is notable as it is known that ulcerative colitis most often begins in the rectum and sigmoid

olon; the situation of the growths in this series therefore appears to ontradict the generally accepted theory that the duration of the colitis s one of the main factors predisposing to malignant change. On the ther hand, the duration of symptoms of the patients supports the impor- ance of the time factor.

The four patients with the site of the growth at the distal part of he bowel were distributed through the series without any unusual dif- erentiating features that might serve to explain the situation of the umour. Characteristic of the entire material, in addition to the com- ination of colitis and cancer, is the low age of the majority of the patients nd the relatively low age of the remainder compared with the usual ncer age. It is possible that this second time factor may be of impor- nce for the unusual situation. The possibility of detecting the tumours y inspection or digital examination of the rectum is, of course, much ss, and the roentgenologic examination of the colon for a malignant owth assumes added importance in the presence of this condition.

The investigation indicates that there is no constant characteristic in the roentgenologic appearances of the colon with ulcerative inflammatory changes and the subsequent development of carcinoma. There are many cases with short, contracted colons, but there are also cases in which the colitic changes had improved, or the colon had returned to normal length and haustration. The material thus also shows that severe changes in the wall due to colitis may heal without fibrosis of the muscular layer and the serosa. This healing, if it occurs, is however no guarantee against malignant degeneration, even though the symptoms of the patient may have also decreased or even disappeared.

SUMMARY

Twenty-six cases of ulcerative colitis with malignant degeneration were investigated; two-thirds of these had advanced inflammatory changes in a chronic stage and one-third showed roentgen evidence of improvement of the colitis. Only 15 % of the growths were situated at the recto-sigmoid junction, 85 % appearing in other parts of the colon.

ZUSAMMENFASSUNG

Die Verfasser haben 26 Fälle von ulceröser Colitis mit maligner Entartung untersucht. Von diesen Fällen hatten 2/3 fortgeschrittene entzündliche Veränderungen chronischer Art, und 1/3 zeigte röntgenologisch Verbesserung der Colitis. Nur 15 % der Tumoren waren am Übergang vom Rectum zum Sigmoideum gelegen; 85 % traten in anderen Abschnitten des Dickdarmes auf.

RÉSUMÉ

Les auteurs ont examiné vingt-six cas de colite ulcérative avec dégénérescence maligne; deux tiers de ces cas avaient des lésions inflammatoires avancées à un stade chronique et un tiers présentaient des signes radiologiques d'amélioration de la colite. Seulement 15 % de ces tumeurs siégeaient à la jonction recto-sigmoïdienne, 85 % étant situées dans d'autres parties du colon.

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GAS MYELOGRAPHIC STUDIES IN SYRINGOMYELIA

by

Gösta Klefenberg and *Georg-Fredrik Saltzman*

Syringomyelia is, from the clinical and symptomatologic aspects, a fairly well-defined disease entity characterized by a dissociated reduction in sensibility with loss of appreciation of painful stimuli and temperature at an early stage, often in combination with atrophy of the hand muscles and segmental pain. It runs a protracted, progressive course.

The pathologic background is not entirely uniform. Glia-proliferation and hydromyelia, or in other words intramedullary cavities filled with fluid are prominent features but the relative proportions between these two components show much variation. In rare cases the glia-proliferation may take the form of true tumours of the glioma and ependymoma type. The signs of syringomyelia are in theory not associated with any special group of pathologic changes but rather to a certain localisation of various forms of destructive changes in the spinal cord.

The intramedullary cavities may attain imposing dimensions particularly in the cervical region but sometimes also in the upper part of the thoracic spine. The spinal cord appears to be much increased in size at the site of these large cavities. We have endeavoured to ascertain by gas myelography whether enlargement of the spinal cord can be demonstrated in clinically typical cases of syringomyelia; films from a

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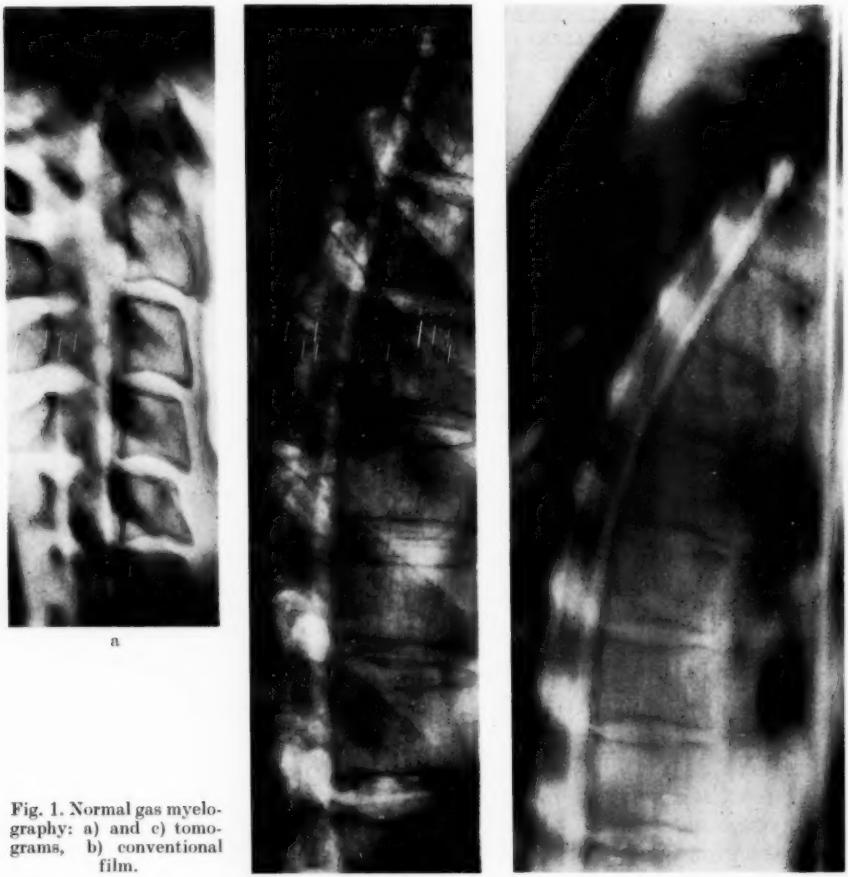

a b c

Fig. 1. Normal gas myelography: a) and c) tomograms, b) conventional film.

gas myelographic study in one such case have previously been published by ODÉN. We have also investigated the possibilities offered by this method for distinguishing between volume increases in the cord due to syringomyelia and other types of intramedullary expansive processes.

The pathologic material consisted of 28 clinically characteristic cases of syringomyelia. All the patients were adults. Our aim was to compare the gas myelograms from these cases with those obtained from cases with no pathologic changes in the spinal cord. Gas myelography is too serious an intervention to be performed in symptom-free subjects without

definite indications. We considered, however, that we had found a usable 'normal material' in a number of patients who had been examined by gas myelography in order to distinguish between rupture of an intervertebral disk in the lumbar region and tumours in the lowermost part of the spinal cord, but only those in whom no tumours could be detected were accepted for investigation. This material consisted of 20 adults, and is called the 'comparison' material. In addition, gas myelograms from 9 patients with intramedullary expansive processes in the cervical and upper thoracic parts of the spine were studied; only those cases in which the whole cord could be examined were included.

The myelographic technique has been worked out by LINDGREN and during the last few years has been combined with tomography.

The conventional films were taken with a film-focus distance of one meter. The magnification varied slightly, of course, according to the size of the patient; the distance from the spinal cord to the film varied, particularly in the lateral views which were obtained with vertically directed rays, but the difference was only 5 cm maximally, and was seldom more than 3 cm, and thus the differences due to magnification were not measurable. At the corresponding tomographic examination the focus-object distance was 95 cm and this meant that the magnification was slightly greater than in the conventional films. Here too, however, the difference as shown in both phantom tests and in comparative films made by gas myelography proved to be so small that, in practice, we were able to compare measurements from conventional films and tomograms without correction. The true measurements are obtained with sufficient accuracy for all practical purposes by multiplying the values obtained in the films by 0.8. All values given in this paper refer to measurements registered in the films.

Comparison material

In the 20 patients composing the comparison material we endeavoured to measure the size of the spinal cord at the level of every other vertebra, starting from the first cervical vertebra downwards. These measurements were carried out in lateral views taken with vertically directed rays. The values may be studied in the appended diagram from which it may be seen that the sagittal diameter of the spinal cord at each level varied within a fairly narrow range. The greatest variations were recorded in the lowermost part of the cord.

The sagittal diameter of the cord decreased gradually in the cervical region. Thus, no increase in the sagittal diameter corresponding to the intumescentia cervicalis could be established. This accords well with the view that it is mainly the transverse diameter that increases at the site

of the latter (RAUBER-KOPSCH). In the uppermost part of the thoracic spine, the sagittal diameter of the cord attained the measurement that was consistently obtained throughout the greater part of the thoracic segment. The thickening of the lowermost part of the cord began at slightly different levels; in the majority of the cases it was distinctly seen at the level of the twelfth thoracic vertebra, and seemed to be manifest at the level of the first lumbar vertebra except in the few cases in which the cord did not reach so far downwards.

Among the individual measurements, one or two deserve special mention because of their significance in comparison with the syringomyelia cases. In none of these cases used for comparison was a sagittal diameter greater than 12 mm measured in the cervical part of the spine. The smallest sagittal diameter obtained in the thoracic spine was 7 mm. There was no relation between the highly variable sagittal diameter of the spinal canal and the sagittal diameter of the spinal cord in this material.

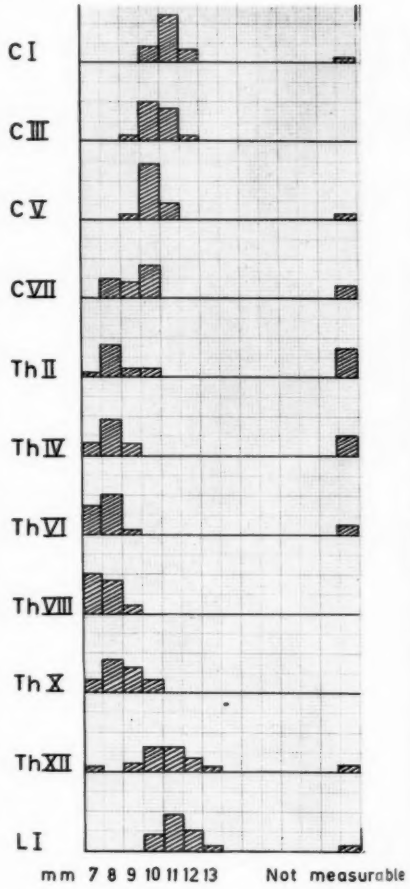

Diagram showing variations in the sagittal diameter at different levels of the normal spinal cord. Number of cases is indicated by the height of the columns, each millimeter square corresponding to one case.

The condition laid down for inclusion of a case in this comparison material was that the spinal cord in its entirety must be readily estimated. However, as may be seen from the diagram, it was not possible to obtain all the measurements desired; in the upper part of the thoracic region, in particular, the conditions were poor for carrying out exact measurements. There is every reason to believe that the use of higher voltages in roentgen diagnosis will be an aid in reducing these difficulties. In the present material, the maximum voltage available was used at least for all views of the lower cervical and upper thoracic regions, but our apparatus did not permit of voltages higher than 100 kV.

Fig. 2. Clinically characteristic syringomyelia. Cervical part of spinal cord enlarged and thoracic part atrophied.

Syringomyelia cases

Of the 28 cases with clinically characteristic syringomyelia in which technically acceptable gas myelography had been performed, 11 were examined in 1952 or later, in other words with a combination of myelography and tomography. In the other 17 cases, only conventional films were taken. As a result of this, the values for the sagittal diameter of the spinal cord could not be recorded as consistently as in the comparison series.

The measurements in the syringomyelia material were regarded as pathologic when they fell entirely outside the distribution recorded at the corresponding level in the comparison series. In 6 cases all measurements obtained lay within these 'normal' variations; in one of the cases, however, the diameter of the cord at the level of the third cervical vertebra was 2 mm greater than at the level of the first cervical vertebra, a finding not obtained in a single case in the comparison series.

In the other 22 cases, at least one, and in most instances several, measurements did not lie within the distribution range of the comparison material. A greater sagittal diameter was found in 14 cases, i. e. in half the material. The examination was combined with a. p. tomograms in some of these cases and it was then found that not only the sagittal but also the transverse diameter was greater than normal. An operation was performed in 8 of these cases. The site of the spinal cord enlargement demonstrated by gas myelography was verified in every instance. The enlargement was found to be due to cavities typical of syringomyelia. It was wholly confined to the cervical spine in 7 cases; it began in the cervical spine and extended down into the thoracic region in 4, while in 3 cases it was entirely limited to the thoracic spine.

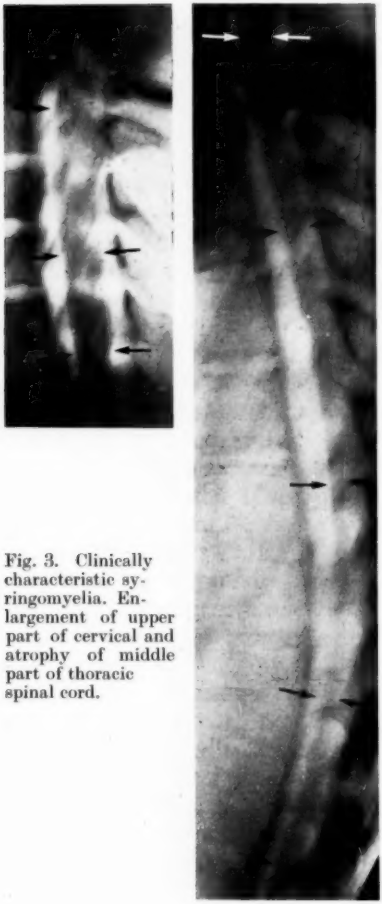

Fig. 3. Clinically characteristic syringomyelia. Enlargement of upper part of cervical and atrophy of middle part of thoracic spinal cord.

Fig. 4. Clinically characteristic syringomyelia. Cord enlarged in cervical region and atrophied in upper thoracic but of normal size in lower thoracic region.

The length of the enlarged segment showed much variation. The longest segment in our material extended for 12 vertebrae and the shortest for 3 vertebrae. In 4 cases, all verified at operation, complete obstruction to the passage of the gas was noted during gas myelography carried out after suboccipital puncture. The length of the enlarged cord could not be estimated in 3 of these cases as the examination was not supplemented with gas myelography after lumbar puncture.

In 9 of the 14 cases with localized increase in size of the spinal cord. the sagittal diameter of the cord below the affected segment lay in all

Fig. 5

Fig. 6

Fig. 5. Clinically characteristic syringomyelia. Advanced atrophy of the cord in lower thoracic area. Cord enlarged from C3 downwards. (Entire spinal canal in lower cervical and upper thoracic regions obliterated.)

Fig. 6. Clinically characteristic syringomyelia. Cord not enlarged anywhere but atrophic throughout the thoracic region.

Fig. 6

the measurements obtained within the range of variation measured in the comparison series. In 5 cases, however, the cord caudal to the enlargement was narrower than at the corresponding level in any of the cases in the comparison material. Atrophy of the spinal cord was obviously present in these cases (Figs. 2 to 5) and was so advanced in 2 of them that the sagittal diameter of the cord did not exceed 3 mm. The atrophic segment was completely localized to the thoracic part of the spine in all 5 cases. In one instance, the cord was extremely narrow in two different segments, with a slightly wider section between them.

In the remaining 8 cases of syringomyelia there was no enlargement of the cord, but at least some part of it had a sagittal diameter which was less than at the corresponding level in any of the control cases (Fig. 6).

In all 8 cases, however, the atrophy was localized, at least partially, t
the thoracic spine. In 2 cases the cord was seen to be narrow in the low
part of the cervical spine and in the areas so affected there were sig
of small volume variations in one or two cases although these were n
of measurable proportions.

Finally, it may be mentioned that among the 13 cases wit
demonstrable atrophy, 8 of the examinations had been combined wit
tomography. Among 15 cases in which atrophy was not established on
3 had been examined by tomography. This suggests that the cases wit
atrophy might have been more numerous than our measurements ind-
cated.

The age distribution in the syringomyelia material is recorded belo
together with the corresponding figures for the comparison series. The
was no great difference between the two groups of cases.

Age	Comparison cases	Syringomyelia cases
20—29 years	3	8
30—39 »	8	7
40—49 »	5	9
50—59 »	2	3
60—69 »	2	1
Total	20	28

Intramedullary expansive processes without signs of syringomyelia

Gas myelograms from 9 adult cases of operatively verified intramedullary expansive processes in the cervical and upper thoracic parts of the spine without signs of syringomyelia were also studied. The main reason why the number of cases is so small is that several patients with processes of this nature had been operated upon after the presence of total obstruction to the passage of gas had been established by gas myelography following suboccipital puncture, and without supplementary gas myelography after lumbar puncture to complete the examination. The spinal cord caudal to the process could consequently not be estimated and these cases were thus not included in our investigation.

The intramedullary expansive processes in this group consisted of 4 cases of ependymoma, 3 of astrocytoma, 1 of carcinoma metastasis, and 1 of arteriovenous aneurysm. None of these processes caused total obstruction to the passage of the gas.

These gas myelograms were studied mainly from the standpoint of the appearance of the cord caudal to the process. In 8 of the cases, the sagittal diameter, wherever it could be measured, varied within the range

Fig. 7. Astrocytoma at the junction of cervical and thoracic regions, filling the entire spinal canal. Cord of normal size caudal to tumour.

found to apply at the corresponding level in the comparison material (Fig. 7). In only one case did we obtain a measurement approaching the lower borderline for the 'normal' variations (6 to 7 mm at the level of Th3). The diameter at the level of the adjoining vertebrae was not less than 7 mm, however.

Conclusions

In the majority of the clinically characteristic cases of syringomyelia in which a technically satisfactory gas myelographic examination could be made, the anatomy of the spinal cord differed from that noted in a series of cases which could with almost complete certainty be regarded as normal. We observed in the syringomyelia cases both enlargements of the cord and regions narrower than in the comparison series. Enlarged segments were noted in the cervical and upper part of the thoracic spine, while the atrophic areas were wholly or predominantly localized to the thoracic region. When both of these changes were present in one and the same patient the enlarged part of the cord was always situated cranially to the atrophic area. Syringomyelia with spinal cord enlargement but without atrophy could not be distinguished from an intramedullary expansive process of some other type. A study of 9 cases of intramedullary spinal cord processes without signs or symptoms of syringomyelia suggested that a combination of enlargement and atrophy may be a characteristic feature of syringomyelia. No change of this nature could, in fact, with certainty be demonstrated in the spinal cord in the cases with intramedullary expansive processes in other forms.

Tomography is a valuable aid when large areas of the spinal cord are to be estimated by gas myelography. Theoretically speaking, a technique based on the use of high voltages ought also to offer considerable advantages. It is especially important in syringomyelia cases, in which the spinal cord lesions are distributed over large areas of the spine, that the conditions for examination should permit a reliable estimation of all parts of the cord. We have not performed myelographic examinations with iodized oil in cases with clinically characteristic syringomyelia. It is obvious, however, that gas myelography must offer better possibilities for studying the spinal cord in cases of syringomyelia than myelography with iodized oil, which only permits examination of the cord in sections and gives films in which the sagittal diameter is often very difficult to measure.

SUMMARY

Twenty-eight patients with clinically characteristic syringomyelia have been examined by gas myelography. The results were compared with 20 normal gas myelograms as well as with observations in 9 cases with intramedullary expansive processes without signs of syringomyelia. The concomitant occurrence of enlargement and atrophy of the spinal cord seems to be characteristic of syringomyelia.

ZUSAMMENFASSUNG

Achtundzwanzig Patienten mit klinisch charakteristischer Syringomyelie sind mit einer Luftmyelographie untersucht worden. Die Ergebnisse wurden mit 20 normalen Luftmyelographien und mit Untersuchungen in 9 Fällen mit intramedullären expansiven Prozessen ohne Anhaltspunkte für Syringomyelie verglichen. Das gleichzeitige Auftreten von Verdickung und Atrophie des Rückenmarkes scheint für die Syringomyelie charakteristisch zu sein.

RÉSUMÉ

Vingt-huit malades atteints de syringomyélie cliniquement caractéristique ont été examinés par myélographie gazeuse. Les résultats ont été comparés avec 20 myélographies gazeuses normales ainsi qu'avec les constatations faites dans 9 cas de tumeur intramédullaire sans signes de syringomyélie. L'existence concomitante d'un élargissement et d'une atrophie de la moelle paraît caractéristique de la syringomyélie.

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OSTEOSCLEROTIC CHANGES IN MYELOMATOSIS

Report of a case

by

Olle Odelberg-Johnson

Bone lesions are demonstrable in about 80 % of all cases of myelomatosis (BAYRD 1947 and HEISER 1952) and are generally described as purely osteolytic without any signs of reaction of the surrounding cells. Three types of lesions are usually distinguished. The most characteristic of these are circumscribed purely destructive lesions of varying size and resembling punched-out cavities. Such defects are usually seen in the cranium, ribs and pelvic bones. Changes of the second type resemble soap-bubble conglomerates of cystic rarefactions. SCHINZ (1952) calls these lesions "die hypertrophische Athrophie mit Wabenzeichnung". The third type (HEISER 1952 and WILLIAMS 1958) resembles osteoporosis. In this form the lesions are sometimes so extensive as to obscure the normal structure of the bone and since they often occur in the spine, compression fractures of affected vertebrae are not rare.

Solitary myeloma is osteolytic and characterized by slow local quiescent growth for several years and sometimes shows a final rapid formation of multiple osteolytic metastases in various bones. The solitary myeloma also occasionally gives rise to sclerosis due, either to a thickening of the trabeculae following roentgen therapy and occasionally after treatment

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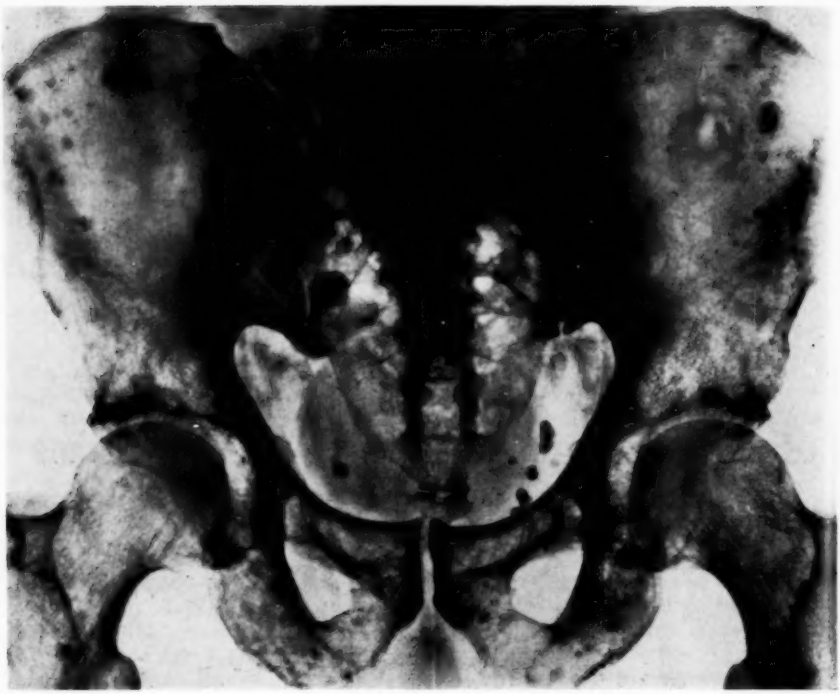

Fig. 1. Pelvis with multiple osteosclerotic changes.

with urethane, or to deposition of amyloid. SCHINZ describes the picture of amyloid deposition: "Das Röntgenbild is durch eine starke, fleckige Knochenzerstörung im Geschwulstinneren und eine intensive endostale und periostale hyperostotische Randreaktion gekennzeichnet. Der erste Eindruck lautet auf Geschwulstmetastase oder Osteomyelitis".

A few cases of myelomatosis with osteosclerosis are on record. In the case described by KRAININ (1949) typical osteolytic lesions were demonstrated in the cranium, femurs, clavicles and right humerus; the left humerus, however, showed massive new bone formation and a pathologic fracture. It was not known whether the fracture preceded the sclerosis. Microscopic examination of a biopsy specimen from the site of the fracture showed abundant plasma cells in the marrow and extensive, cortical new bone formation with spicules. In KOHLER's (1950) case the pelvic bones showed osteolytic as well as sclerotic changes. BIONDETTI (1952) described a case with purely sclerotic lesions in the humerus, ribs and

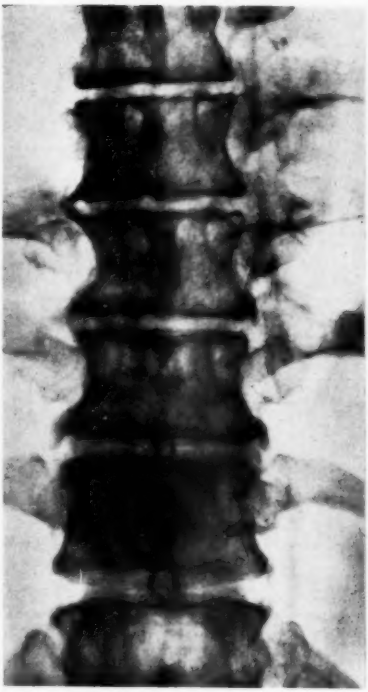

Fig. 2. Lower thoracic vertebrae. Circumscribed osteosclerotic lesions resembling metastases, due to myelomatosis.

Fig. 3. First to third lumbar vertebrae.

vertebrae, purely osteolytic lesions in the skull and mixed lesions in the femurs and pelvic bones.

In the cases referred to above the sclerotic changes thus occurred together with osteolysis. Microscopic examination of the sclerotic tissue was not performed in the two last mentioned cases.

One case of questionable myelomatosis with sclerotic lesions only, was described by SHARNOFF (1954). Such changes were present in the lower lumbar and sacral vertebrae, and compression fractures were observed in the 5th and 6th thoracic vertebrae. A biopsy specimen from the sternum revealed 'atypical plasma cells throughout marrow. The osseous tissue was markedly sclerotic, extremely dense, with small lacunae and no osteoblastic activity'. The presence of plasma cells in huge amounts in the blood and the clinical course of the disease were

regarded by the author as suggestive of plasma cell leucemia. No definite decision was made between this diagnosis and myelomatosis.

WIDEBÄCK (1956) observed a woman with mottled sclerosis in the skull and the thoracic and lumbar vertebral column. Roentgen examination 16 months later showed no further changes. Serologic examination and smears of the bone marrow from the sternum suggested myelomatosis, while needle biopsy from the iliac crest showed marked myelofibrosis and myelosclerosis but no changes characteristic of myelomatosis.

The following case report is of a patient with purely sclerotic bone lesions due to myelomatosis.

Case report

Male, aged 51, admitted to hospital in December 1955 with neurologic signs (impaired sensibility of feet). Flaccid paresis of the arms and legs developed, and from January 1956 he was confined to bed. During the first year of the disease the clinical picture was dominated by fatigue. During the second year the patient lost weight, the ESR increased, and he had transient diabetes. Lumbar puncture showed a protein increase to between 100 and 200 mg per 100 ml but a normal number of cells. Provisional diagnosis of polyradiculoneuritis (Guillain-Barré syndrome) was made. Electrophoresis of the serum in February 1955 had shown increased alpha globulins and decreased albumin. This abnormal pattern persisted during the patient's later stay in hospital. The serum calcium, serum phosphorus, alkaline phosphatases, haemoglobin and red blood cell count were repeatedly normal.

In June 1956 *roentgen examination* revealed sclerotic changes resembling metastases in the vertebrae and pelvic bones. No such changes had been observed on roentgen examination made four years earlier for uretherolithiasis. Further clinical and roentgen examination produced no evidence of any primary tumour in the lungs, prostate or thyroid gland. Sternal and iliac puncture showed abundant plasma cells in the bone marrow. A biopsy specimen was removed from one of the roentgenologically demonstrated lesions in the iliaca crista. Gross inspection showed osteosclerotic bone tissue and microscopic examination revealed abundant plasma cells.

The patient died in October 1956.

Post mortem examination: The eleventh thoracic vertebra was found to contain a large, pale, circumscribed osteosclerotic area. The first and second lumbar vertebrae contained small lesions of similar appearance.

Microscopic examination: Many coarse bone-trabecules at site of these lesions. Between the trabecules were abundant plasma cells. There was cancellous bone outside the osteosclerotic areas containing fine bone trabecules and the bone marrow was rich in cells. Apart from a moderate diffuse increase in plasma cells this bone marrow contained ordinary bone marrow cells in normal proportion. Diagnosis: Myelomatosis (Prof. F. Linell).

It may be mentioned that the patient never received roentgen therapy. After the discovery of the sclerotic lesions he had, however, been treated with urethane for a short time.

In 1958 WILLIAMS wrote that "one may state dogmatically that if t ere is any sclerotic reaction at the margin of an osteolytic focus this i not due to a plasmocytoma or myeloma. The only new bone formation I have seen in myeloma either follows irradiation or is due to callus f mation with a pathologic fracture". The observations presented above, h wever, show that osteosclerosis may occur in myelomatosis and that it may be the only lesion demonstrable in the roentgenogram.

SUMMARY

A case of myelomatosis is reported in which roentgen examination revealed progressive and widespread, circumscribed osteosclerosis resembling metastases but no evidence of osteolysis. Histologic examination of sclerotic parts of the skeleton repeatedly showed characteristic myelomatous changes.

ZUSAMMENFASSUNG

Ein Fall mit einer Myelomatosis wird berichtet, bei dem die Röntgenuntersuchung eine progressive und multiple circumskripte Osteosklerose aufwies, welche Metastasen ähnelte, aber keinerlei Osteolyse zeigte. Wiederholte histologische Untersuchungen sklerotischer Knochenteile zeigten charakteristische myelomatöse Veränderungen.

RÉSUMÉ

Les auteurs présentent un cas de myélomatose où l'examen radiographique a révélé une ostéosclérose circonscrite progressive et disséminée, ressemblant à des métastases, mais sans signe d'ostéolyse. L'examen histologique de parties sclérosées du squelette a montré, à plusieurs reprises, les lésions caractéristiques de la myélomatose.

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EEG AND ECG IN CEREBRAL ANGIOGRAPHY WITH SODIUM DIATRIZOATE (HYPAQUE) AND METYL-GLUCAMINE DIPROTRIZOATE (MIOKON)

by

Torgny Greitz and *Stuart Weiss*

Despite the synthesis of increasingly less toxic contrast media, complications still occur in cerebral angiography. The classical investigations of BROMAN and OLSSON (1949) showed that the water-soluble contrast media cause cerebral damage by breaking through the blood-brain barrier. However, very little has been written about the clinical demonstration of the effect of the contrast media upon the brain. FOLTZ, THOMAS, and WARD (1952) studied the effect of Diodrast in man and animals by electroencephalography (EEG) during cerebral angiography. A similar study on patient undergoing cerebral angiography was made by INGVAR (1957). MELIN (1952) and KIRSTEN et coll. (1957) performed EEG following angiocardiography. GREITZ (1956) suggested that the electrocardiographic (ECG) changes seen during cerebral angiography were due to a direct action of the contrast medium upon the vasomotor-reactive regions of the brain. This assumption was verified by LINDGREN and TÖRNELL

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(1958). SJÖGREN (1958) used ECG to compare the relative merits of certain new contrast media in cerebral angiography.

FOLTZ et coll. observed marked EEG changes in their patients. They used relatively large doses (15 ml of a 35 % solution) of Diodrast which must be regarded as a fairly toxic agent. INGVAR was unable to demonstrate any EEG alterations with 10 ml Umbradil 35 % which, although chemically identical with Diodrast, is supposed to possess less toxicity. In recent years two new contrast media have come into use for cerebral angiography—sodium or methylglucamine diatrizoate (Hypaque) and sodium or methylglucamine diprotrizoate (Miokon). Clinical experiences with these media indicate that they are less toxic than Diodrast or Umbradil. It is of interest, therefore, to examine the effect of the new media as shown by EEG and ECG. As either procedure could be a sensitive detector of potentially injurious actions of the contrast media upon the brain, a comparison between any changes should be of value.

Technique. Six ml Hypaque or Miokon 30 to 35 % were injected into the internal or common carotid artery. In this study Hypaque was used as the sodium salt and Miokon as the methylglucamine salt. Up to a maximum of four injections were made in each subject, both contrast media being employed. Percutaneous puncture was performed in all cases and the injection was completed within 1 to 2 seconds.

Needle electrodes were inserted intracutaneously into the right and left frontal, vertex, and temporal areas of the scalp. Another electrode was inserted over the precordium. All electrodes were earthed to eliminate any interference from the roentgen equipment. Bipolar EEG and ECG recordings were made simultaneously, prior to, during, and after injection of the media. The period of the injection was noted on the record. All subjects were questioned as to whether they experienced any discomfort during the procedure.

Material. The subjects used in this study were chosen at random from those on the neurological and neurosurgical registers of Barnes Hospital, St. Louis, who were to have percutaneous carotid angiography as a routine diagnostic procedure. Four patients were males and three females, their ages ranging from 7 to 66 years. The tentative clinical diagnoses were cerebral concussion with encephalography, subdural hematoma, tuberculous meningitis, hypertensive encephalopathy, epilepsy, cerebrovascular insufficiency and tumor. At angiography, pathologic changes were found in the case of subdural hematoma and in the case of tuberculous meningitis. The latter showed bilateral narrowing of the carotid siphon.

Results

EEG and ECG were recorded during 24 injections in 7 patients, 2 of whom had bilateral angiography. The injections were made 20 times into the internal and 4 times into the common carotid artery. Eleven injections were performed with Hypaque, 13 with Miokon. No complications occurred. No EEG changes were recorded during any injection. However, during 17 out of 24 injections ECG changes were observed, appearing either as a slight slowing in the pulse rate (7 injections), an increase in the pulse rate (6 injections), or bradycardia followed by tachycardia (4 injections). No ECG changes were observed on three injections with Hypaque and four with Miokon. No qualitative or quantitative difference was found between Hypaque and Miokon in any tendency to provoke ECG changes or discomfort in the subjects. No increase in the severity of the reactions was noted with repeated injections.

Discussion

The results are in agreement with those of INGVAR who observed no EEG changes when Umbradil was used as the contrast medium. The changes commonly observed by FOLTZ et coll. following Diodrast injections may have been due to one of two possibilities: that Diodrast is a more toxic agent than either Umbradil, Hypaque, or Miokon, or that these authors used larger doses. Since the amount of contrast medium employed in our studies was sufficient to obtain satisfactory results, we have not felt justified in increasing it to make quantitative comparison with the former study.

Since the bradycardia and/or tachycardia also occurred following injections into the internal carotid artery above the bifurcation, it is believed that coincident stimulation of the carotid sinus or carotid body as a complication may be eliminated (GREITZ). The experimental investigations of LINDGREN and TÖRNELL have also definitely shown that the bradycardia which occurs in cerebral angiography derives from the vasomotor-reactive regions of the brain and not from the carotid sinus. Thus, the bradycardia at least (and likely all the ECG changes) indicates a direct action of a contrast medium upon the brain. It should be remembered that while both ECG and EEG are bioelectric phenomena, they have entirely different origins. Of the two, ECG is the more sensitive indicator of the threshold effects of injection. Since EEG is a record of the spontaneous activity of the cerebral neurones, the fact that no changes were noted in this study is a substantiation of the relative safety of the

small doses and less toxic media used nowadays in cerebral angiography. The value of ECG in detecting potential danger is exemplified by the case of SJÖGREN, in which marked ECG changes were recorded in a patient who developed hemiplegia in connection with carotid arteriography.

SUMMARY

EEG and ECG were carried out in 7 patients during 24 injections for cerebral angiography with sodium diatrizoate and methylglucamine diprotrizoate. No EEG changes were recorded, but ECG changes were frequently seen. It is concluded that ECG is on the whole a more sensitive indicator of potential danger to the brain ensuing from the injection of a contrast medium.

ZUSAMMENFASSUNG

EEG (Elektroencephalogramme) und EKG (Elektrokardiogramme) wurden an 7 Patienten während 24 cerebralangiographischer Injektionen mit Natriumdiatrizoat und Methylglukamindiprotrizoat ausgeführt. Keine EEG-Veränderungen wurden registriert. EKG-Veränderungen sah man jedoch häufig. Es wird gefolgert, dass das EKG ein empfindlicher Indikator einer potentiellen Gefahr für das Gehirn nach einer Kontrastmittelinjektion ist.

RÉSUMÉ

Des EEG et des ECG ont été enregistrés au cours de 24 injections pour angiographie cérébrale faites sur 7 malades avec le diatrizoate de sodium et le diprotrizoate de méthylglucamine. Il n'y a pas eu de modification de l'EEG, mais les modifications de l'ECG ont été fréquentes. Les auteurs en concluent que l'ECG est dans l'ensemble un indicateur plus sensible du danger potentiel pour le cerveau de l'injection d'un moyen de contraste.

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FROM THE X-RAY LABORATORY OF PHILIPS' GLOEILAMPENFABRIEKEN, EINDHOVEN, HOLLAND

QUALITY AND CHOICE OF POTTER BUCKY GRIDS

II. Application of the criterion of quality to various types of grids

by

J. G. Bonenkamp and *W. Hondius Boldingh*

It was explained in part I how the contrast-improving quality of a grid may be efficiently evaluated. It was shown that this could be done in a simple way by means of a graph in which contrast is plotted as a function of the exposure time with the voltage as a parameter, at constant density and tube load. The method by which this quality graph may be obtained was also indicated in part I. In the present part, the results of measurements relating to thirteen grids, with characteristics given in Table 1, will be discussed. Measurements without a grid are also recorded.

The measurements were performed in water phantoms of 10, 20 and 30 cm, the contrast objects were 1 cm air and 2 mm Al. The voltage was varied between 50 and 200 kV, the added filter on the tube being 0.5 mm Al + 0.05 mm Cu. The exposure times are based on $D = 1.0$, at a tube load of 4 kW, whilst the contrast obtained is related to a film gradation of $\gamma = 2$. In order to register the differences as clearly as possible a wide beam (36 mm diameter at a FFD of 90 cm) was used in all the measurements.

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Table 1

Characteristics of the grids used in the measurement of contrast-improving quality

Numbering in sequence of increasing lead weight	Lamellae			Ratio = height / distance	Number of lines/cm	Lead content		Particulars
	Thickness in mm	Height in mm	Distance in mm			cm²/cm²	mg/cm²	
0	—	—	—	—	—	—	—	No grid
1	0.05	1.1	0.32	3.4 : 1	27	0.015	170	Al interspaced
2	0.05	2 × 1.1	0.35	2 × 3.1 : 1	25	0.027	310	Cross-grid (about 2 × No. 1)
3	0.05	2.5	0.37	7 : 1	24	0.030	340	
4	0.03	2.5	0.22	11 : 1	40	0.030	340	
5	0.06	2	0.29	7 : 1	29	0.035	390	
6	0.08	2	0.37	6 : 1	24	0.038	430	
7	0.05	3.5	0.38	9 : 1	23	0.040	460	
8	0.03	3.5	0.23	15 : 1	38	0.040	460	
9	0.05	2 × 2.5	0.37	2 × 7 : 1	24	0.060	680	Cross-grid (2 × No. 3)
10	0.05	4.5	0.31	15 : 1	28	0.063	720	
11	0.06	4.5	0.31	15 : 1	27	0.073	830	
12	0.06	6	0.39	15 : 1	22	0.079	900	
13	0.07	4.5	0.30	15 : 1	27	0.085	960	

1. Lead content and ratio

The measurement results for a water phantom of 30 cm and a contrast object of 2 cm air are plotted in Diagram 1, from which it may be seen that the various grid characteristics virtually do not intersect throughout the voltage range.

As we shall see later (Diagram 3), the same applies to the other water phantoms and contrast objects, with the exception of the relatively unimportant case of a 10 cm water phantom and 1 mm Al. The upper lines refer to the grids which are best in regard to contrast-improving capacity, as was explained in part I. As the grids are numbered from 1 to 13 in the order of increasing lead content per cm² and, generally speaking, a higher numbered grid is situated higher in the graph, it can be seen that

> grids with the highest lead content give
> the greatest improvement in contrast.

A high lead content is usually associated with a high ratio. In order to be able to decide whether the quality is determined by the lead content or by the ratio, the contrast at one specific exposure time (line A—B in Diagram 1) was plotted as a function of the lead content (Diagram 2a) and as a function of the ratio (Diagram 2b).

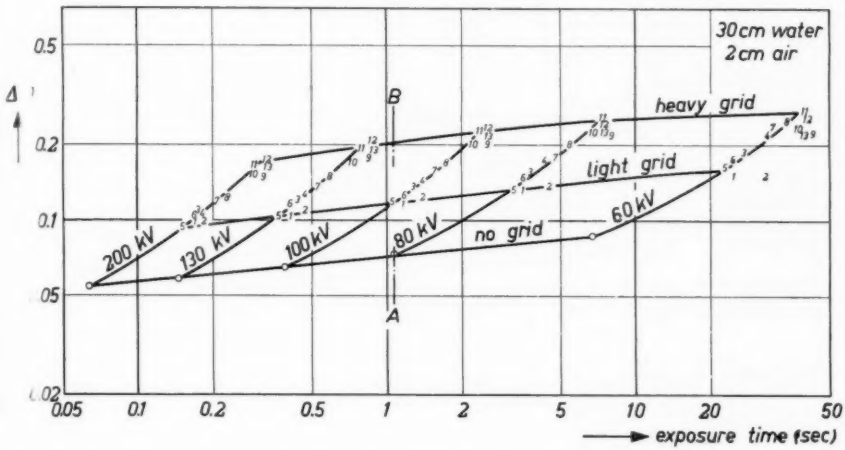
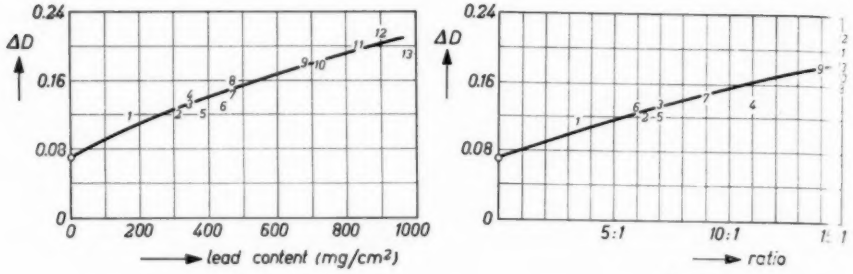


Diagram 1. Contrast ΔD as a function of exposure time from 60 to 200 kV. Bottom line, no grid; second line, lightest grid; top line, heaviest grid. Numbers 1 to 13 correspond to the grids used in the measurements, numbered in sequence of their lead content.

By means of these diagrams we arrive at the conclusion that the ratio is not of primary importance. This appears from the fact that two grids with the same lead content viz. Nos. 3 and 4, and 7 and 8, respectively, virtually coincide although they have different ratios viz. 7 and 11, and 12 and 15, respectively. The five grids Nos. 8, 10, 11, 12, and 13, on the other hand, with different lead contents, differ fairly considerably in 'quality' although they have the same ratio, 15. An example of the reverse has not been observed.

The lead content can therefore be considered as a measure of the contrast-improving effect of a grid, but only if the lead is applied in an effective manner, with a well chosen relationship between the height and thickness of the lamellae. This latter requirement is readily recognized if it is borne in mind that a given lead content in the form of a lead plate would provide a very poor grid. In such a case, the scattered radiation is absorbed to an almost equal degree as compared with a high ratio grid, whereas the transmission of the primary radiation is reduced to a very small percentage in comparison with a normal grid; this causes the characteristic graph to be situated at nearly the same height as the line 'without grid', with the additional unfavourable difference that a considerable shift to the side of the long exposure times will occur (Cf. LINDBLOM 1934).

The significance of lead content and ratio will be discussed more



a b

Diagram 2. Contrast ΔD as a function of: a) lead content, and b) ratio.

in detail in part III but we conclude for the present that, for the sake of discrimination it is better to speak of heavy and light grids than to use the ratio as a quality factor.

A study of Diagram 2a may cause the question to be raised whether 'quality' can be increased infinitely by increasing the lead content. This is obviously not the case. First of all there is a theoretical limit, corresponding to the 'ideal' grid ($T_s = 0$, $T_p = 100\ \%$, $C = C_p$). Furthermore, it is true that when the lead content increases, the stray radiation is still more suppressed, but this is also the case with the primary radiation. It is quite clear therefore that the prolonged curve in Diagram 2a would show a maximum contrast improvement at a certain (unusually high) lead content above which no gain in contrast would be obtainable with this phantom thickness and contrast object. It is, however, not certain that such an excessively heavy grid can be made with an acceptable ratio and number of lamellae per cm. We shall return to this subject in part III.

2. Grids with aluminium interspacing and cross-grids

It is interesting to note that grid No. 1, and cross-grid No. 2, both with aluminium interspacing, and cross-grid No. 9 with normal, organic interspacing, do not produce any special improvement in contrast as compared with other grids with a corresponding lead content. In Diagram 2a these grids occupy a place in accordance with their lead content (Cf. LEDIN 1952).

3. Influence of phantom thickness and contrast medium

The results of 6 different combinations of phantom thicknesses and contrast-objects are recorded in Diagram 3. Each combination is, for the sake of clearness, represented by four lines, the bottom line indicating

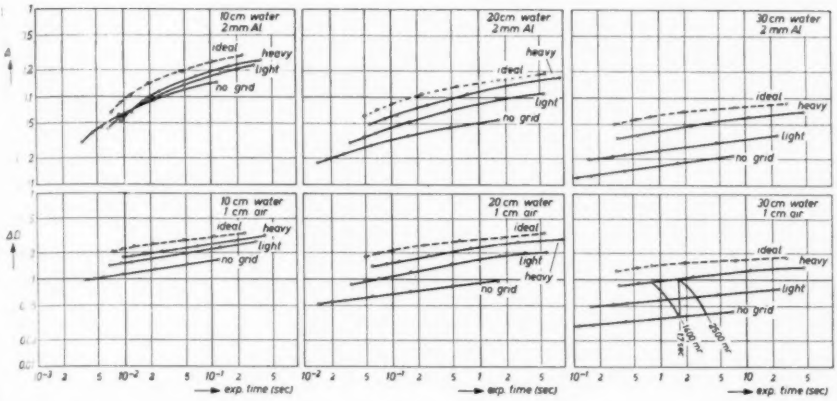


Diagram 3. Contrast as a function of exposure time (4 kW d. c., density 1.0, gradation 2.0, wide beam) of 2 mm Al and 1 cm of air in water phantoms of 10, 20 and 30 cm thicknesses from 50 or 60 kV to 200 kV. Inherent tube filter 2 mm Al, added filter 0.5 mm Al + 0.05 Cu.

exposures without grid, the next two lines the lightest and the heaviest grids, respectively, and the dotted top line the case mentioned under heading (1), i. e. an ideal grid that removes all scattered radiation without loss of primary radiation, giving the limit of the theoretically obtainable contrast. Five of these combinations including the one dealt with in the mentioned section (30 cm of water and 2 cm of air) have the same character, and the conclusions made before that the heaviest grids yield the greatest improvement in contrast therefore hold good. This consequently obtains not only independently of the voltage (50 to 200 kV) but also of phantom thickness (10 to 30 cm of water) and contrast object (Al or air).

One exception is represented by the case of a 10 cm water phantom with Al as contrast object. The lines intersect, and it is found, quite unexpectedly, that at high voltages it is precisely the exposures without grid which yield the best contrast, whilst at low voltages the use of a grid is to be preferred. This case is however of academic interest only; the differences are negligible and this agrees with practical experiences, namely that grids are not necessary in the case of 'bone structure' in thin phantoms.

4. Dose measurements

In view of the increasing interest in the dose received in radiography, measurements were carried out on all three water phantoms in the whole range of 50 to 200 kV, with 0.5 mm Al + 0.05 mm Cu filter, to

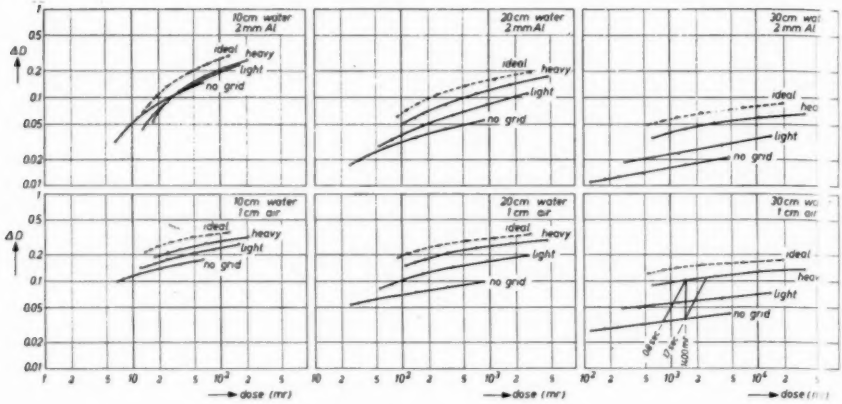


Diagram 4. Contrast as a function of dose, free air, measured at 50 cm distance from the focus, other conditions being equal to those in diagram 3.

ascertain what free-air-dose is applied at a distance of 50 cm to obtain the density 1.0 in the film when the various grids are used, and without grid. In the case of a 20 cm water phantom, filters of 1 mm Al and 0.5 mm Al + 0.2 mm Cu were also used. For the sake of simplicity, the increase of the dose at the surface of the phantom due to backscatter. which is larger with higher voltages and which may vary between 10 and 20 %, has not been taken into account. We have also not included the volume dose in our considerations.

These measurements enable us to obtain a good insight into the interconnection between contrast, exposure time, voltage and dose (Cf. WACHSMANN 1952).

Two isodose lines have been drawn for this purpose in the grid lines for a 30 cm water phantom with air contrast (Diagram 3).

It will be seen that when a heavier grid is chosen in order to obtain better contrast, at a given exposure time, not only the voltage must be raised (e. g. from 75 to 110 kV) but also a higher dose (2 500 mr instead of 1 400) is applied. It will moreover be found that with the same heavy grid and at the same dose, a shorter exposure time is obtained (0.8 instead of 1.6 sec) when the voltage is still further raised, up to 125 kV, and one is satisfied with a slightly smaller gain in contrast.

A more complete idea of the effect of Bucky grids on the dose is obtained when plotting the contrast as a function of the dose instead of as a function of the exposure time (Diagram 4).

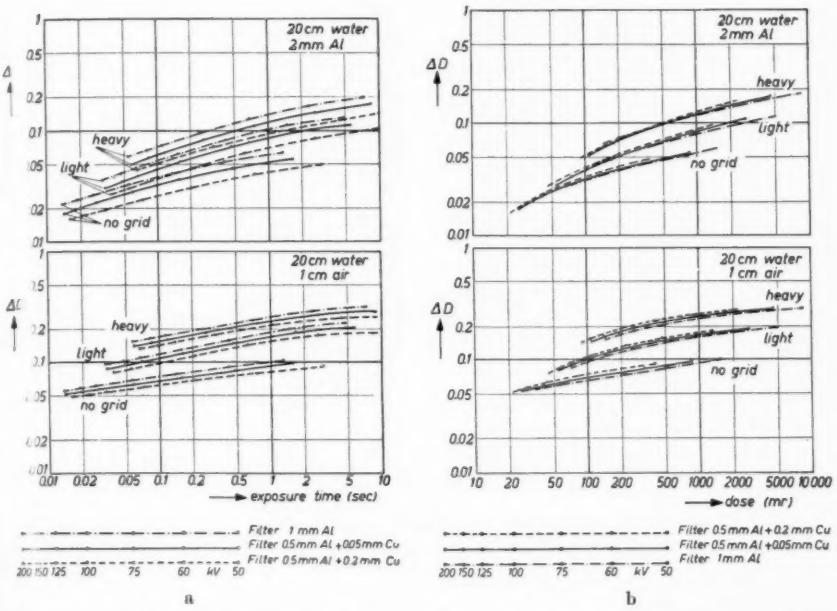


Diagram 5. Contrast as a function of: a) exposure time and b) dose, free in air, with various added filters.

In this case too, the 10 cm water phantom with aluminium contrast forms an exception; the three lines again intersect so that in the case of thin objects with 'bone structure' the best contrast is obtained with the same dose at high voltages without a grid and at low voltages with a grid. This phenomenon again is of minor importance because with these objects the dose applied is small in any case, and, a fortiori, the differences in dose in the case of different grids are practically negligible.

5. Filters

In order to study the influence of various filters on exposure time and dose, in addition to the measurements given in Diagrams 3 and 4, with a filter of 0.5 mm Al + 0.05 mm Cu, similar measurements have been carried out with a thinner filter (1 mm Al) and a thicker one (0.5 mm Al + 0.2 mm Cu) (See Diagram 5, inherent tube filter 2 mm Al). In (b) where the contrast is given as a function of the dose, it can be seen that in the entire voltage range of 50 to 200 kV the three corresponding lines almost coincide but are shifted slightly in their own direction.

In the case of roentgenograms of a given object, for which the voltage is so chosen that $D = 1.0$ is obtained with a given dose, almost the same contrast is achieved with different filters. A thinner filter then calls for a higher voltage and a shorter exposure time.

The well-known rules for the use of filters in diagnostic radiology (heavier filters for higher voltages) achieve a decrease in dose with a thicker filter at unaltered voltage, at the expense of loss of contrast and longer exposure time.

6. High voltage technique (up to 200 kV)

It would seem worthwhile to summarize quantitatively the effect of high voltages on contrast, exposure time, and dose, on the basis of the above measurement results with a wide beam. The gain factors may be evaluated from Diagrams 3 and 4 as recorded in Table 2.

Table 2

Gain factors	125 to 150 kV		150 to 200 kV		125 to 200 kV	
Phantom thickness	10 cm	30 cm	10 cm	30 cm	10 cm	30 cm
Exposure time	1.6	1.9	1.7	2.2	2.7	4.2
Dose	1.2	1.4	1.15	1.45	1.4	2.0

It appears that the gain in the range 125 to 200 kV is considerable for the exposure time used with thick objects.

As regards the loss of contrast, a study of Diagram 5 will disclose the recognized fact that the loss of contrast of an 'air' object is considerably smaller than that of a bone object, and that this is especially the case with thin objects. This explains why the advocates of the use of high voltages are chiefly met with in the field of lung radiography. In addition to the gain in exposure time and dose there is the greater loss of contrast in the bone structure causing the well known 'transparency' of the ribs, which in the opinion of some radiologists counterbalances the (slighter) loss of contrast in the air-filled lung tissues.

Our general rule also applies in the case of lung radiography at high voltages, namely that the heaviest grids yield the best contrast.

Our graphs give no evidence for the contrast conditions when using heavy contrast media such as iodine and barium. In these cases the loss of contrast due to high tension is often less important than the gain in exposure time. Here again the heaviest grid is to be recommended.

It should not be forgotten, on the other hand, that the blurring due to intensifying screens may be greater at higher tensions than at lower

o ies (Cf. FROST 1957). This may be compensated more or less by the gain in visibility due to reduced blurring caused by movement and improvement in contrast.

SUMMARY

Measurements on 13 different grids reveal that their contrast-improving capacity is determined mainly by their lead content. Grids with aluminium interspacing and cross grids do not form exceptions to this rule. The relation between contrast, free-air-dose, and filters, is discussed, especially at high voltages. In part III, to be published shortly, an analysis of the fact that the lead content is the main criterion of grid quality will be made and the relation between lead content, ratio and number of lamellae per cm will be dealt with in some detail. Criteria for the choice of grids for various fields of application will be indicated.

ZUSAMMENFASSUNG

Messungen an 13 verschiedenen Gitter zeigen, dass ihr Kontrastverbesserungsvermögen hauptsächlich durch den Bleigehalt bestimmt ist. Gitter mit Aluminiumspalten und Kreuzgitter bilden keine Ausnahme von dieser Regel. Die Beziehungen zwischen Kontrast, Dosis und Filter werden besprochen, insbesondere bei hohen Spannungen. Im folgenden dritten Teil werden Betrachtungen über die Tatsache angestellt, dass der Bleigehalt das Hauptkriterium der Gitterqualität ist; die Beziehungen zwischen dem Bleigehalt, dem Verhältnis sowie der Anzahl der Bleilamellen per Quadratzentimeter werden ziemlich eingehend behandelt. Hieraus kann man dann Kriterien für die Wahl von Gittern für verschiedenartige Anwendungsbereiche aufstellen.

RÉSUMÉ

Des mesures sur 13 grilles différentes montrent que leur pouvoir d'amélioration du contraste dépend surtout de leur teneur en plomb. Les grilles à lamelles intercalaires d'aluminium et les grilles croisées ne font pas exception à cette règle. Les auteurs examinent les relations entre le contraste, la dose dans l'air et les filtres, surtout pour les tensions élevées. Dans la partie III qui suivra, les auteurs montreront que la teneur en plomb est le critère principal de la qualité des grilles et étudieront en détail les relations entre la teneur en plomb, le rapport et le nombre de lamelles par centimètre. Des critères pour le choix des grilles pour différents domaines d'utilisation seront indiqués.

REFERENCES

(For list of references, see part I, Acta radiol. 51 (1959), p. 489.)

FROM THE PHYSICS DEPARTMENT (CHIEF PHYSICIST: L. A. W. KEMP) OF THE LONDON HOSPITAL, WHITECHAPEL, LONDON, ENGLAND

PHYSICAL ASPECTS OF ROENTGEN THERAPY USING WEDGE FILTERS

II. Analysis of wedge field dosimetry

by

M. Cohen

In the first part of this paper (COHEN 1959) some examples were given of isodose charts of wedge pairs, with and without an additional normal field. These are typical of the 30 or so charts prepared for the 4 wedge filters in regular use at the London Hospital, and a large number — perhaps the majority — of patients and lesions can be made to fit one or other of these charts. But the problem of the 'non-standard' lesion still remains, and, although individual treatments can always be planned with wedge as with normal fields, the number of variables is so large that some systematization is necessary if optimum results are to be achieved. At the same time, the analysis to be described has proved valuable in indicating which arrangements are best fitted for 'standard' treatments, and even in pointing to the kind of wedge fields that ought to be produced in the future.

Statement of the problem. The variables to be investigated are shown in Fig. 1. Two wedges are set so that they make an angle θ (usually 60 to 90°). The thick ends always face towards the apex of θ: sometimes they

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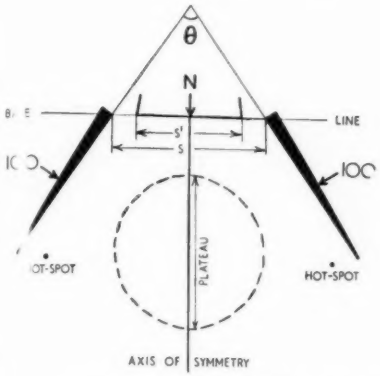

Fig. 1. General arrangement of wedge pair and normal field, showing the variables featured in the analysis.

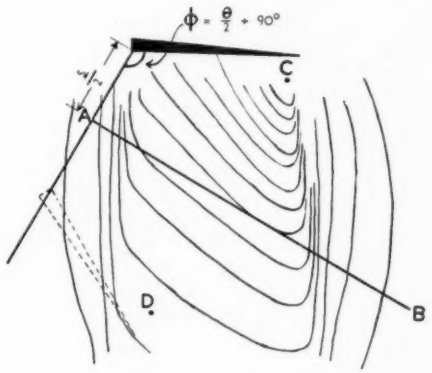

Fig. 2. Method of obtaining 'plateau curves' (Diagram 1): AB = axis of symmetry of Fig. 1, origin at A. Hot-spot dose = dose at C (1 cm from wedge) + dose at D (mirror image of C in AB) + contribution from normal field assessed from its isodose curves.

are contiguous, but in general they are separated by a distance s. In this gap is applied the normal field, of width s'. It will be assumed throughout that the normal and wedge fields are applied from the same side of the body, and furthermore that the normal field is applied along the line joining the thick ends of the two wedges — the so-called 'base line'.

The incident dose to each wedge is taken as 100, that for the normal field as N. 'Incident dose' in both cases has its usual meaning: the central axis surface dose with full backscatter. N can take any value from zero upwards, a common value being 50.

In addition to the variables θ, N, s and s', concerned with the addition of wedge fields, there are three variables associated with the individual wedge filter: the wedge size, the slope of the isodose lines (as defined in part I) and the shape of the isodose lines. All three have been subjected to analysis, as will be described later.

Throughout this analysis it is assumed that the incident radiation is as specified in part I, i. e. 250 kV, 1 mm Cu + 1 mm Al filter, 1.74 mm Cu HVL, and that the wedge filters are those previously described. If this is not so, the numerical values deduced will have to be adjusted; this situation will be discussed in part III.

The factors listed above are the independent variables of the problem; the dependent variable is the dose distribution for the combined field. Ideally, one should have a set of isodose curves for each field arrangement, but the preparation of a very large number of isodose charts would

be extremely laborious and in any case it would be difficult to compare them in a quantitative way. Fortunately isodoses are unnecessary for the analysis, since the essential features of each distribution may be extracted in the form of three factors: the mean percentage tumour dose, the position of the region of uniform dose distribution, relative to the defined 'base line', and the maximum skin dose. These factors may be deduced without actually drawing out the isodose chart of the arrangement concerned.

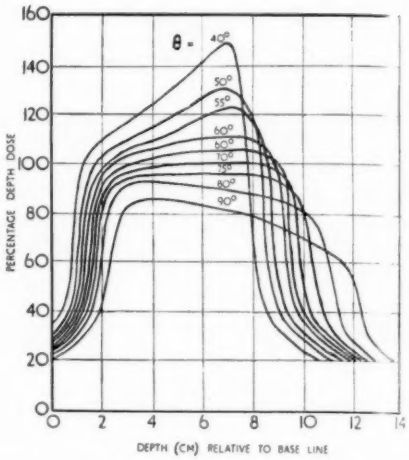

Diagram 1. 'Plateau curves' for 6 × 8 cm wedge pair, $\theta = 40°$ to $90°$, $s = 6$ cm, $N = 0$.

Preparation of 'plateau curves'. When two wedges, or two wedges plus a normal field, are set up as in Fig. 1, the essential feature of the resulting dose distribution will be a region of more-or-less uniform dose in the centre of the configuration. This is shown as a broken circle in Fig. 1, though in general the region will not be circular in shape. Above and below this region the dose falls rapidly, while to either side the dose builds up to a maximum near the thin edge of each wedge. The key to the whole pattern is the dose distribution along the axis of symmetry of the arrangement. The dose along this axis may be obtained by drawing a line $A\,B$ in the appropriate position on the isodose chart of the wedge (Fig. 2) and reading off the dose levels at the points of intersection.

Curves of axial distributions for a 6 × 8 cm wedge (no normal field) for various values of θ, s being 6 cm throughout, are given in Diagram 1. It is seen that, starting from the base line, there is a steep rise at first, followed by a more-or-less horizontal section (corresponding to the region of uniformity) which will be termed the 'plateau', and finally a fairly rapid fall.

Definitions of plateau position and mean tumour dose. In Diagram 1 it is seen that as θ increases from 40° to 90°; the height of the plateau, in terms of percentage depth dose, decreases, while the plateau itself occurs at increasing depths below the base line. Only when $\theta = 75°$, in this example, is the plateau absolutely flat; for other angles there is a gradual rise to a peak. This does not matter for clinical purposes, provided the peak is not too sharp. In order to assess the effect of peak sharpness

in practice, the limits of the plateau must be defined, even though the definition is necessarily an arbitrary one: for our purpose we take the plateau as that part of the curve in which the dose is within 10 % of the peak value. Clearly, the sharper the peak the smaller will be the extent of the plateau lying within these limits. Since the plateau is supposed to coincide with the tumour during treatment, the mean tumour dose is taken simply as the mean plateau dose, i. e. the peak dose minus 5 %. The dose over the whole plateau is therefore, by definition, uniform within the limits $\pm$ 5 %. In practice the uniformity may be rather better than this, especially when the plateau is fairly flat, since the 'mean' as just defined is not a weighted mean in the strict mathematical sense. The mean tumour dose so defined has been found to agree closely, in individual cases, with that estimated by independent inspection of the full isodose chart.

Addition of a normal field. The curves of Diagram 1 refer to two wedge fields only. Any one of these curves may be used as the starting point for a new family of curves showing the effect of adding a normal field. Diagram 2, for example, refers to the addition of an 8 $\times$ 6 cm normal field to a 6 $\times$ 8 cm wedge pair at $\theta = 60°$, $s = 6$ cm. N has been assigned values 0, 25, 50, 75 and 100. In this example a normal field dose of 25 (where each wedge incident dose is 100) is sufficient to cancel out the peak on the right of the $N = 0$ curve, giving a flat plateau, while if N is raised above 25 a peak appears on the left side and this gets steeper as N is increased.

It was seen in Diagram 1 that (with this particular wedge pair) if θ is greater than 75° the peak of the $N = 0$ curve already lies to the left. In such cases the addition of a normal field can only make the peak steeper and steeper — no flat plateau position is passed through as in the case of $\theta = 60°$, $N = 25$. Hence, in certain circumstances (e. g. small values of θ) the addition of a normal field may improve the dose distribution, or at least, not appreciably worsen it, while in other cases (e. g. large values of θ) the normal field only serves to introduce a non-uniform element into an already uniform picture provided by two wedges alone. In the latter circumstances the justification for the use of the third field lies solely in the reduction of the maximum skin dose.

The 'hot-spot factor'. This is defined as the ratio of the maximum skin dose to the mean tumour dose. Although the true maximum in any configuration occurs on the face of the wedge applicator, in practice this rarely coincides with the skin, since a layer of wax usually lies between them. Indeed, it was for long the custom at the London Hospital to insist on at least 1 cm of wax between the applicator and the skin, in order to

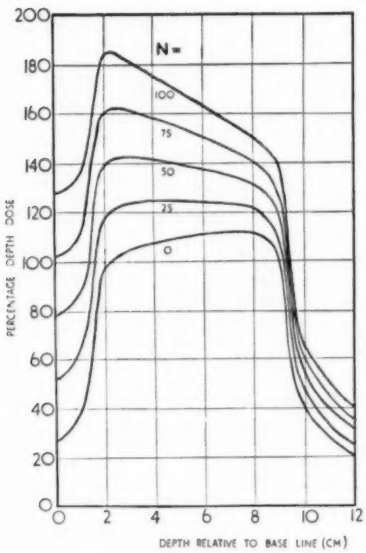

Diagram 2. 'Plateau curves' for 6 × 8 cm wedge pair, $\theta = 60°$, $s = 6$ cm, plus 8 × 6 cm normal field, $N = 0$ to 100.

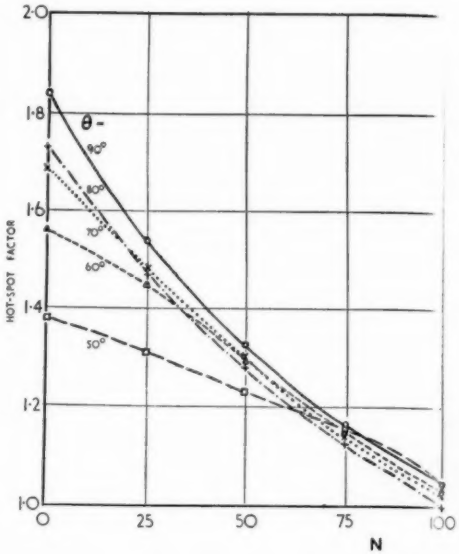

Diagram 3. Hot-spot factor versus N, conditions as for Diagram 2.

spare the skin (this point is examined critically later) and it was therefore postulated that the maximum skin dose occurred 1 cm from the applicator face and 5 to 8 mm in from the edge of the field, depending on the wedge size. This convention was adopted at the onset of the present work, and although the 1 cm wax requirement has now been abandoned, it was not considered worthwhile to alter the convention. As in Fig. 1, therefore, the hot-spot factor refers to a point 1 cm from the wedge. It will be shown later that at the wedge itself the factor is increased by only 2 to 4 %.

In Diagram 3 hot-spot factors are plotted against normal field dose for different values of θ, other factors being as in Diagram 1. It is seen that when $N = 0$ the hot-spot factor is much greater for large angles θ than for small: only at 50° is it clinically possible to dispense with the normal field. As N is increased the hot-spot factor decreases rapidly, and for $N = 50$ it is about 1.3 for all values of θ. This is satisfactory clinically, and Diagram 3 may well give the impression that, provided N is at least 50 it does not matter what angle the wedges are set at: a large angle may safely be used if it is more convenient to do so. However, before reaching a decision the location of the region of uniform dose must also be considered.

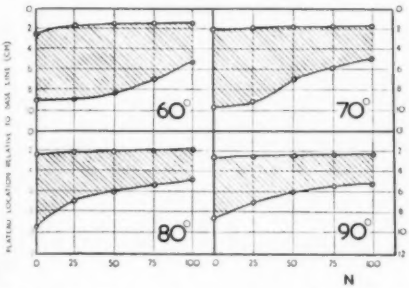

Diagram 4. Plateau location versus N, conditions as for Diagram 2 ($\theta = 50°$ omitted).

Diagram 5. Plateau depth (full lines) and hot-spot factor (broken lines) versus N, for 6×8 cm wedge pair, $\theta = 60°$, $s = 4$, 6 and 8 cm, plus 8×6 cm normal field.

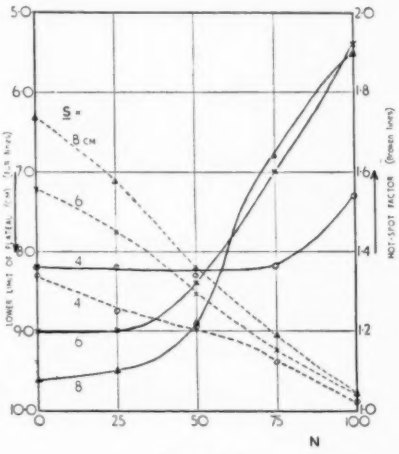

Diagram 5.

The plateau location. The limits of the plateau have already been defined in relation to the peak of the axial curve. These limits may be plotted as in Diagram 4, which refers to the same conditions as Diagram 3 except that $\theta = 50°$ is omitted. The upper limit of the shaded portion represents the beginning of the plateau: it lies about 2 cm below the base line for all values of θ and N. But the lower limit of the plateau depends on both θ and N, such that the larger the normal field dose the nearer the termination of the plateau approaches the base-line, and furthermore, the larger the angle θ the more quickly this effect sets in.

Analysis of the individual variables

As already explained, any two or three field wedge arrangement may be assessed in terms of the mean percentage tumour dose, the plateau location and the hot-spot factor. Of these, the mean percentage tumour dose is unimportant in so far as it only determines the duration of exposure in an actual treatment, but does not influence the choice of one configuration as against another. For the latter purpose the plateau location and the hot-spot factor are the determining factors. Although on occasion a deliberate choice may be made to restrict the depth of penetration of the plateau region into the body, as a general rule the difficulty is to obtain sufficient penetration rather than vice versa, so that a configuration which gives a deep plateau penetration will be considered more useful than one which yields only a small penetration, provided

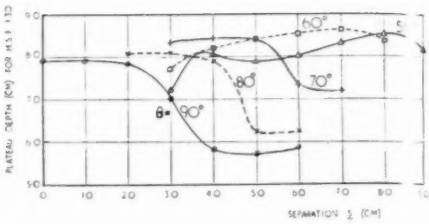

Diagram 6. Plateau depth versus s (parameter θ) for 6 × 8 cm wedge pair plus 8 × 6 cm normal field, when HSF = 1.30.

that the hot-spot factors are equal in the two cases. Usually, however, a deep plateau is associated with a large hot-spot, so the problem is to find the configuration which yields the best compromise between these factors.

If the problem concerns a known, individual patient, it is a straightforward matter to pick a number of possible configurations which will fit the site concerned and to choose the one which gives the most satisfactory combination of hot-spot factor and plateau location. This will be illustrated in more detail later. Meanwhile, however, we are concerned with the more general problem — that of describing the role played by each variable, with a view to assessing the relative importance of the parameters, to formulating general rules for wedge field additions, to finding the most suitable configurations for 'standard' use, and to improving the design of individual wedge filters.

Strictly speaking, if an individual variable is to be studied, all other factors must be kept constant. It is found, however, that the results provided by this means are so cumbersome that little useful information can be extracted. A simple modification, however, allows the effect of each variable to be expressed in readily understood terms. The method is to allow the normal field dose, N, to 'find its own level' in satisfying certain fixed conditions applying to the other variables. Supposing, for example, the variable s were being studied. Curves of hot-spot factor versus N, and of the lower limit of the plateau versus N (i. e. Diagram 3 and half of Diagram 4) are plotted, on a common scale, for a number of values of s, the other variables being constant (Diagram 5). (As previously shown, the upper plateau limit tends to remain constant and in any case is comparatively unimportant.) It is now only a question of reading off from each pair of curves, associated with a particular value of s, the value of plateau depth corresponding to an arbitrary value of hot-spot factor. Except where specifically mentioned, the value of hot-spot factor used for this purpose throughout the analysis is 1.30, a value corresponding to that aimed at in therapy. For example, in Diagram 5, the broken curve for $s = 8$ cm gives HSF = 1.30 when $N = 59$. The

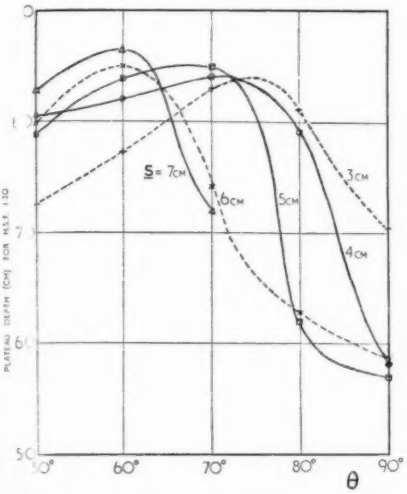

Diagram 7. Plateau depth versus θ (parameter s) for 6×8 cm wedge pair plus 8×6 cm normal field, when HSF = 1.30.

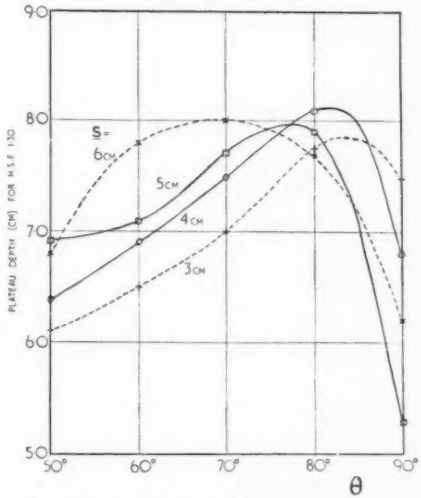

Diagram 8. Plateau depth versus θ (parameter s) for 5×5 cm wedge pair plus 5×5 cm normal field, when HSF = 1.30.

corresponding full curve ($s = 8$ cm) shows that at this value of N the plateau penetration is 8.2 cm. Finally the plateau depths so obtained from each pair of curves are plotted against s, with parameter θ, as in Diagram 6.

The justification for this procedure is not only that it yields a clear picture of the effect of each variable (as in Diagram 6) but that it also corresponds closely to the realities of treatment. Every other variable except N is — at least within a fairly narrow range — governed by the geometry of the site to be treated. But N may be (and is) varied at will to suit the requirements of the case, and the precise value of N does not matter in the least provided the resulting dose distribution is satisfactory. We are therefore justified in regarding N as entirely 'at our disposal' in studying the other variables.

The variables θ and s. These are the most important pair of variables in treatments with a given set of wedges. Their role has already been illustrated to some extent in Diagrams 1 to 6, but is brought out more clearly in Diagrams 7 to 9. These have been deduced by the method just described, using a constant value of 1.30 for the hot-spot factor. Diagrams 6 and 7 (for the 6×8 cm wedge) are clearly interchangeable by cross-plotting, and, similarly, graphs corresponding to Diagram 6 for the 5×5 cm and 8×10 cm wedges may be obtained from Diagrams 8 and 9, respectively, but are not reproduced.

Diagrams 7 to 9 show that, for a given wedge and given value of s, there is a value of θ which yields the best wedge performance in terms of plateau penetration. If this value of θ is exceeded the plateau depth drops rapidly. The optimum value of θ increases as s is decreased, but there is one value of θ which is near optimum for the whole range of s values considered. This is about 80° for the 5 × 5 cm wedge, about 65° for the 6 × 8 cm wedge and (probably) about 50° for the 8 × 10 cm wedge. These are the values of θ which should therefore be chosen for the production of standard sets of isodose charts. Unfortunately, in the case of the 8 × 10 cm wedge, 50° is too small an angle for the majority of sites, and the use of 60° results in a serious loss of plateau penetration unless the separation s can be reduced below its usual value of 8 cm. Clearly this wedge, in its present form, is not ideal.

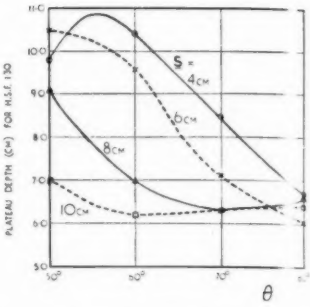

Diagram 9. Plateau versus θ (parameter s) for 8 × 10 cm wedge pair plus 10 × 8 cm normal field, when HSF = 1.30.

Diagram 6 indicates that there is no optimum value of s; rather the plateau penetration remains approximately constant over a range of s, but falls if this range is exceeded. The critical value of s, at which this fall sets in, is (for the 6 × 8 cm wedge) 2 cm at $\theta = 90°$, 4 cm at 80°, 5 cm at 70° and 8 cm or more at 60° and 50°. Combining the information contained in Diagrams 6 and 7, it is clear that there are over a dozen combinations of θ and s which will give a plateau penetration within the range 7.9 to 8.7 cm. For a given patient, some of these may be regarded as alternative arrangements in that they will fit the site concerned and — assuming maximum plateau penetration below the skin is required — the final choice will depend on which arrangement gives the least 'dead space' between the base line and the skin. The same arguments apply to the other two wedges, except that the optimum range of plateau penetration is a little less (7.5 to 8.1 cm) for the 5 × 5 cm wedge (Diagram 8) and a little more (9.5 to 10.7 cm) for the 8 × 10 cm wedge (Diagram 9). In practice, in the case of the 8 × 10 cm wedge it may not always be possible to work under optimum conditions, though even here judicious application of Diagram 9 may be rewarding. For example, at 60°, if it is possible to decrease s from 8 to 6 cm, a gain of 2.5 cm in plateau penetration is obtained. If the criterion of hot-spot factor is taken as 1.40 instead of 1.30, the curves of Diagram 9 are raised by an average of 6 or 7 mm but their shapes are not essentially altered.

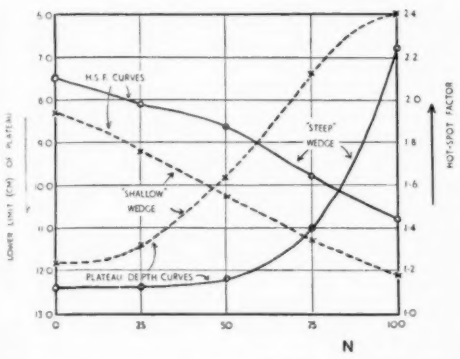

Diagram 10. Plateau depth and hot-spot factor versus N, for 8×10 'steep' (50° slope) wedge (full lines) and 8×10 cm 'shallow' (40° slope) wedge (broken lines) plus 10×8 cm normal field.

The wedge size. The size of the wedge is not usually a variable in the sense that, for a given patient, there is a choice between the four standard wedges available. That decision is usually governed by the geometry of the treatment site, especially as the field length in the non-wedge direction is different in the four applicators. Also, it tends to be assumed that a larger wedge automatically gives a greater plateau penetration. Although, as shown above, this is indeed the case when each wedge is used at optimum, the differences are not so large as to rule out the possibility of obtaining a more favourable distribution with a smaller wedge where the site geometry is such that (1) the larger wedge cannot be used in a favourable configuration and (2) a smaller field length in the non-wedge direction can be accepted. This possibility has been looked into (the details are omitted here) and it has been shown that in such cases the smaller wedge will frequently give a greater plateau penetration since non-optimum geometry for the larger wedge may be optimum for the smaller (compare Diagrams 7 to 9). Even when the required field length in the non-wedge direction is 10 cm, it may be better to mask down the 6×15 cm wedge to 6×10 cm rather than use the 8×10 cm wedge. (The question of masking down a wedge will be considered in part III of this paper.)

The width of the normal field. Hitherto it has been assumed that a standard normal field is always used in conjunction with a given wedge pair, e. g. 8×6 cm normal with 6×8 cm wedges. Usually there is little or no choice in this matter, the normal field depending on the applicators available, bearing in mind that it is usually desirable that all three fields should have the same length in the non-wedge direction. However, in theory there is no limitation to the width s' of the normal field — it may be greater than, equal to, or less than the wedge separation s (see Fig. 1). For example, a 10×5 cm normal field could be substituted for the 10×8 cm field usually employed with 8×10 cm wedges. The advantage of the narrower field is that it contributes less radiation to the hot-spot region and therefore tends to decrease the hot-spot dose;

the disadvantage of the narrower field is that it has lower percentage depth-doses than a bigger field and so tends to decrease the mean tumour dose. Whether the resulting hot-spot factor (i. e. hot-spot dose/mean tumour dose) is increased or decreased is a matter for investigation.

Such an investigation has been carried out, by the method already described, for a number of combinations of θ and s. In general it appears (details are omitted here) that the wider field is advantageous, but the differences are only marginal, i. e. an increase of a few millimetres in plateau penetration for a given hot-spot factor. At small values of θ and s, however, the hot-spot region might fall within the geometrical beam of the larger field so that the narrower applicator would then be preferable, although it must be added that in such circumstances it is usually possible to dispense with the normal field altogether.

Variables concerned with wedge design

The variables so far considered relate to the choice of geometrical and other conditions for combinations of wedge fields, where the wedges themselves are assumed given and unalterable. The two variables which follow are of no immediate interest in the choice of configurations for therapy, but are important as guides to the design of future wedge filters in deep roentgen therapy.

The wedge slope. The slope of the wedge isodose lines (as defined in part I of this paper) is important in so far as it largely determines the type of dose distributions obtained with combined field arrangements, and the associated hot-spot factors. In general, the steeper the wedge slope the greater the hot-spot of the individual wedge and so the greater the hot-spot factors in three-field arrangements.

Most of the information which follows has been derived from a comparison of the two existing 8 × 10 cm wedges — the 'steep' (50° slope) wedge, produced first but rejected as unsuitable for therapy, with a hot-spot of 217 %, and the 'shallow' (40° slope) with a hot-spot of 170 % (see Fig. 3 of part I). An attempt has also been made to apply the data so derived to the 5 × 5 cm and 6 × 8 cm wedges.

Diagram 10 shows plateau depth and hot-spot factor curves for the two 8 × 10 cm wedges for a typical arrangement, i. e. $\theta = 60°$, $s = 8$ cm. It is clear that the steeper wedge gives a deeper plateau penetration, but this is accompanied by higher hot-spot factors, and this statement is generally true for all values of θ and s. To obtain equal hot-spot factors, the steep wedge requires a normal field dose of N' and the shallow wedge of N, where $N' > N$. Suppose, in Diagram 10, a match is made for the

condition $N = 0$, corresponding to a hot-spot factor of 1.94 and a plateau depth of 11.8 cm for the shallow wedge. To obtain a hot-spot factor of 1.94 with the steep wedge, N' must equal 35, the plateau depth then being 12.3 cm, i. e. 0.5 cm greater than for the shallow wedge. The steep wedge therefore appears advantageous. However, a hot-spot factor of 1.94 is quite unacceptable in clinical practice, so a new comparison must be made on a more realistic basis.

It will be noticed that the general level of hot-spot factor is much higher in Diagram 10 than was the case with the 6×8 cm wedge, so that it is difficult to work on a basis of a 'standard' HSF of 1.30 unless N (or, more particularly, N' for the steep wedge) is greater than 100. While there is no inherent reason why N should not rise above 100, in practice it is found that the dose distributions so obtained bear less and less of the attributes of wedge fields. In the present instance a valid comparison can, however, be made on the basis of HSF = 1.40.

From Diagram 10 the plateau depths are 8.1 cm (shallow wedge) and, by a slight extrapolation, 6.0 cm (steep wedge). A similar comparison has been made over a wide range of values of θ and s, and it is found that only for small values of θ and s (e. g. 50° and 6 cm) does the steep wedge retain its initial advantage, and even then only by millimetres. For more usual configurations the shallow wedge has a decisive advantage: indeed it is probable that a further small decrease in the wedge slope would have been advantageous (see also Diagram 9).

However, the evidence derived from the apparent advantage of the steep wedge at HSF = 1.94 in Diagram 10 cannot be entirely neglected. The reason for this advantage is that the plateau depth of the steep wedge remains practically unchanged between $N' = 0$ and $N' = 50$, so that no adverse effect on plateau depth results from a normal field dose of only 35. This is precisely the situation pertaining to the 5×5 cm wedge: the dosage table for this wedge (to be explained below) shows that, for nearly all the configurations listed, the plateau depth remains virtually constant from $N = 0$ to $N = 50$, in some cases even beyond. However, in marked contrast to the 8×10 steep wedge, this constancy is associated, at the $N = 50$ level, with hot-spot factors which are clinically acceptable, i. e. 1.40 or less. If, therefore, a less steep 5×5 cm wedge were produced, treatments could be given with smaller values of normal field dose than are used at present (perhaps even zero) but there would be no advantage in terms of the plateau depth; on the contrary, Diagram 10 points to a slight disadvantage.

If the same argument is applied to the 6×8 cm wedge (Table 1) it is found that for some configurations (particularly those at small θ) no advantage would be obtained by decreasing the wedge isodose slope, but for others a decrease would be advantageous. We conclude that the

Table 1

Dosage table for 6×8 cm wedge pair plus 8×6 normal field

θ	s	Normal field					
		0			25		
		T. D. %	Plateau depth (cm)	H. S. factor	T. D. %	Plateau depth (cm)	H. S. factor
50°	5 cm	139	4.8—7.9	1.29	149	3.7—7.8	1.2
	6 cm	126	4.8—8.1	1.38	136	3.4—8.0	1.2
	7 cm	114	4.7—8.4	1.48	125	2.8—8.3	1.3
	8 cm	105	5.0—8.8	1.57	115	3.1—8.7	1.4
	9 cm	95	4.5—9.0	1.71	105	2.1—9.0	1.5
60°	4 cm	133	4.1—8.2	1.34	145	2.4—8.2	1.3
	5 cm	122	3.8—8.4	1.42	132	1.5—8.4	1.3
	6 cm	108	2.6—9.0	1.56	118	1.7—9.0	1.4
	7 cm	101	3.9—9.3	1.62	109	1.9—9.2	1.5
	8 cm	92	3.4—9.6	1.74	102	1.9—9.5	1.5
70°	3 cm	129	2.1—8.4	1.38	140	1.1—8.3	1.3
	4 cm	121	2.5—8.7	1.43	131	1.3—8.5	1.3
	5 cm	109	2.0—9.1	1.54	121	1.5—8.9	1.4
	6 cm	96	2.2—9.7	1.69	111	2.0—9.4	1.4
	7 cm	89	2.5—9.7	1.72	103	2.2—8.8	1.5
80°	2 cm	129	1.4—8.4	1.40	144	1.0—8.1	1.2
	3 cm	117	1.6—8.9	1.48	131	1.4—8.4	1.3
	4 cm	105	1.5—9.6	1.52	123	1.4—8.4	1.3
	5 cm	96	2.0—9.2	1.64	115	1.9—6.9	1.4
	6 cm	90	2.4—9.4	1.73	107	2.3—7.0	1.4
90°	0 cm	143	1.2—7.9	1.30	160	0.8—7.4	1.2
	1 cm	128	1.1—8.3	1.40	146	0.8—7.6	1.2
	2 cm	119	1.1—8.7	1.40	138	1.0—7.1	1.2
	3 cm	109	1.7—8.4	1.46	127	1.4—6.8	1.2
	4 cm	98	1.8—8.2	1.58	117	1.9—6.2	1.3

present slope of this wedge is near optimum — probably a few degrees on the high side.

For a given wedge size there is clearly an optimum wedge slope which leads to the most satisfactory relationship between plateau penetration and hot-spot factor when used in field combinations. The present analysis does not provide sufficient information for a precise determination of the optimum wedge slope, but a useful guide appears to be as follows: the optimum wedge slope is the largest slope which permits the formulation of a considerable number of three-field configurations such that, in reducing the hot-spot factor to 1.30 from its initial value at $N = 0$ the plateau depth curve remains sensibly constant. If ways can be found of reducing the hot-spot of the individual wedge filter for a given wedge slope, the optimum wedge slope will then rise.

d e (N)								
50			75			100		
T. D. %	Plateau depth (cm)	H. S. factor	T. D. %	Plateau depth (cm)	H. S. factor	T. D. %	Plateau depth (cm)	H. S. factor
60	1.2—7.8	1.17	171	0.9—7.8	1.12	189	0.8—7.7	1.04
47	1.5—8.0	1.23	159	1.3—8.0	1.16	179	1.3—7.6	1.05
35	1.5—8.3	1.30	149	1.4—8.2	1.19	170	1.2—7.5	1.06
26	1.4—8.6	1.35	141	1.3—8.4	1.22	166	1.2—5.9	1.05
19	1.8—8.9	1.40	138	1.7—7.5	1.22	158	1.5—5.7	1.08
55	1.1—8.3	1.21	170	1.0—8.2	1.12	192	1.0—7.3	1.02
45	1.2—8.3	1.24	163	1.2—7.9	1.13	186	1.2—6.2	1.00
35	1.6—8.4	1.29	154	1.6—7.0	1.15	174	1.5—5.4	1.04
25	1.7—9.0	1.35	144	1.7—7.1	1.21	165	1.7—5.4	1.06
19	1.9—8.9	1.36	137	1.8—6.8	1.19	157	1.7—5.5	1.04
57	1.0—7.9	1.19	179	1.0—7.0	1.06	200	0.9—5.5	0.98
48	1.2—8.1	1.22	169	1.2—7.0	1.08	191	1.2—5.2	0.98
40	1.4—8.0	1.24	159	1.3—6.5	1.11	181	1.3—5.0	1.00
29	1.9—7.4	1.30	148	1.9—6.0	1.14	168	1.8—5.0	1.02
20	2.0—7.6	1.32	139	1.9—6.0	1.16	158	1.8—5.3	1.03
61	0.9—7.6	1.18	182	0.9—6.5	1.07	204	0.8—5.3	0.97
50	1.4—7.3	1.21	170	1.3—6.2	1.09	192	1.2—4.9	0.98
42	1.4—6.6	1.17	162	1.3—5.4	1.04	184	1.2—4.4	0.94
34	1.8—5.8	1.21	153	1.7—5.1	1.08	173	1.7—4.6	0.97
24	2.2—6.2	1.28	143	2.1—5.5	1.13	163	2.1—4.8	1.00
79	0.7—6.8	1.15	200	0.5—5.9	1.08	222	0.4—4.9	1.02
66	0.7—6.5	1.13	187	0.6—5.3	1.03	209	0.6—4.5	0.94
59	0.9—5.9	1.09	172	0.8—5.8	0.98	203	0.8—4.0	0.89
46	1.4—5.8	1.13	168	1.4—5.2	1.02	186	1.3—4.6	0.92
37	1.9—5.2	1.17	157	1.8—4.7	1.04	178	1.7—4.8	0.93

The shape of the wedge isodose lines. This variable is the least amenable to numerical analysis, yet is of great importance in that it bears on what has just been discussed: the relationship between the wedge slope and the hot-spot in a given wedge. The wedge filters used in this analysis and described in part I, were deliberately designed so as to have straight isodose lines with fairly sharp 'noses'. No other completed isodose chart is available for comparison; yet during the trial period preceding the production of the final designs, isodose lines were plotted which were distinctly curved, and it is known that such lines are associated with lower hot-spots than straight lines. In order to provide a basis for comparison, we have therefore drawn a purely hypothetical set of isodose curves for a 6 × 8 cm wedge, by retaining the existing points of intersection and slope of each line at the central axis, but otherwise curving

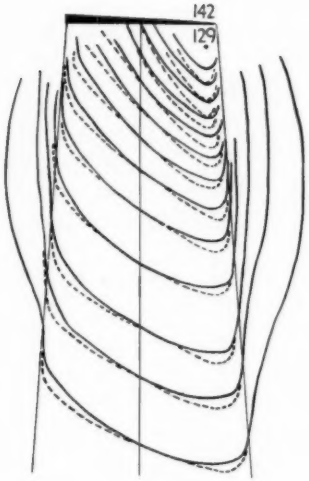

Fig. 3. 6 × 8 cm 'curved' wedge isodose lines (full lines) compared with 'straight' isodose lines (broken lines). The hot-spot figures refer to the 'curved' wedge.

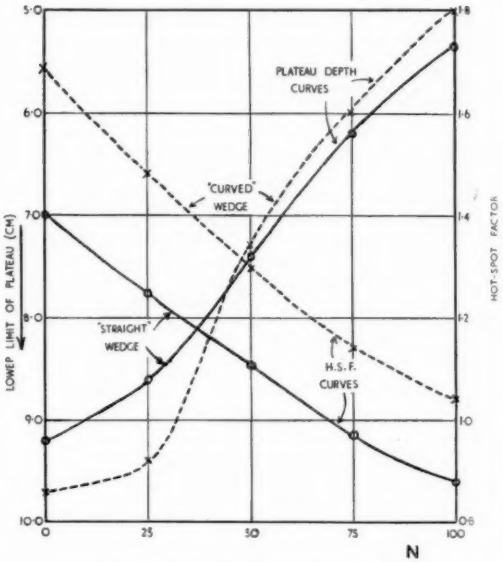

Diagram 11. Plateau depth and hot-spot factor versus N, for 6 × 8 cm 'straight' wedge (full lines) and 'curved' wedge (broken lines) plus 8 × 6 cm normal field.

the lines so that they are less steep on the side of the central axis towards the 'nose' and steeper on the other side. The curves are shown in Fig. 3 (full lines) together with the existing curves (broken lines). The 'new' wedge will be referred to as the 6 × 8 cm 'curved' wedge, and the existing one as the 6 × 8 cm 'straight'. The hot-spot for the curved wedge has been obtained by extrapolating to the surface the depth-doses along a line passing through the nose of each isodose line; the result is 142 %, compared with 155 % for the straight wedge. The extrapolated figure is, of course, open to some error, but there is no doubt that it will be appreciably lower than 155 %, and the present comparison is only intended as a rough guide. Although the isodose curves of Fig. 3 are hypothetical, there is no doubt that a wedge giving such curves could be designed without difficulty: it is mainly a question of transferring some copper from the thin side of the filter to the thick side.

In Diagram 11 a comparison is made between the curved and straight 6 × 8 cm wedges, for $\theta = 70°$ and $s = 6$ cm. The plateau depth is seen

t be somewhat greater at $N = 0$ for the straight wedge but the curves c ss over at about $N = 50$. This seems to be a typical pattern, though t e details vary for different combinations of θ and s; the difference b tween the two plateau depth curves is rarely more than 1 cm. (The u per limit of the plateau is in every case almost coincident for the two w dges.) On the other hand, there is a marked and persistent difference i the hot-spot factor curves — the values for the straight wedge are h her, for every combination of θ and s. In Diagram 11, for a common h -spot factor of 1.30, the plateau depth is 7.2 cm for the straight wedge a l 8.9 cm for the curved. In every case examined the curved wedge is found to be advantageous, though not usually by such a wide margin as in this example. There is clearly a case for the production of curved iso lose lines in future wedge filters.

Applications of the analysis

Applications of the analysis just presented fall into three groups:

1. As already stated, principles for the slope and shape of isodose lines for future wedges are elucidated.

2. Diagrams 6 to 9 provide a guide to the radiotherapist as to which combinations of θ and s give the most satisfactory dose distributions for each wedge. Full isodose charts may be plotted out for some of them for use as standard arrangements. In this connection, however, it must be borne in mind that the points plotted in the diagrams are referred to a constant value (1.30) of hot-spot factor, rather than to round values of normal field dose. For example, in Diagram 7, the point marked for 60°, 7 cm, is obtained when $N = 58$. Though not impossible, it is inconvenient and laborious to combine isodose curves when the normal-field incident dose is 58 % of the wedge-field incident dose, so in this case the field addition would be made with $N = 50$. The hot-spot factor is thereby increased to 1.35, but this is still within the clinically permitted range.

3. Particularly for arrangements not covered by the standard isodose charts, is it useful for the radiotherapist to have as much information as possible in tabular rather than graphical form. Dosage tables have therefore been prepared for each of the three main wedges. (The 6 × 15 cm wedge is used rather infrequently, and its isodose chart is so similar to that of the 6 × 8 cm wedge that the latter may be used with only small corrections.) Only the table for the 6 × 8 cm wedge is reproduced (Table 1), the others being similar but less detailed. For each combination of θ and s, information is given for five values of N, i. e. 0, 25, 50, 75 and 100. Interpolation for intermediate values of N, if required, is readily carried

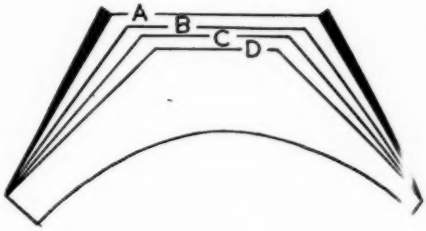

Fig. 4. Alternative arrangements for treatment of a patient with 6 × 8 cm wedge pair plus 8 × 6 cm normal field (see Table 2).

A—$\theta = 60°$ $s = 6.0$ cm
B—$\theta = 70°$ $s = 5.0$ cm
C—$\theta = 80°$ $s = 4.2$ cm
D—$\theta = 90°$ $s = 3.5$ cm

out. The information given consists of: the mean percentage tumour dose, the upper and lower limits of the plateau, relative to the base line, the hot-spot factor. These data are sufficient for treatment to be carried out, without the full isodose chart having to be plotted.

Not every arrangement given in Table 1 is suitable for therapy, in the sense that the hot-spot factor is clinically acceptable. But it is just as important to know when a proposed arrangement is not acceptable as to have full information for permitted configurations. In practice the tables enable a rapid assessment to be made of the relative merits of a number of alternative arrangements for treating a given lesion. In this connection, however, the variation of the 'dead space', between the base-line and the skin, with the different arrangements must be taken into account.

An example of the use of the dosage tables is given in Fig. 4. Here a wax mould is shown on a skin outline; the gaps at the bottom between the skin and the face of the jig arise from the fact that the outline shown is actually the mid-plane of the treatment volume and in other, parallel, planes the body extends outwards and prevents a closer approach to the patient at the base of the arrangement. Previously the standard method of treating this site, with 6 × 8 cm wedges, would have been to use $\theta = 60°$, $s = 6$ cm, $N = 50$. However, various other arrangements are possible, as shown in Fig. 4 and in Table 2. The data in Table 2 is selected from Table 1, (interpolating linearly for s where necessary) with a 'depth under skin' column added. It is seen that, in fact, $\theta = 80°$, $s = 4.2$ cm, $N = 25$ would probably be a better arrangement, although in this example the margin is not wide.

The hot-spot factors in Table 1 are all calculated, as mentioned earlier, on the assumption that 1 cm of wax is placed between the skin and the applicator at the hot edge. This was formerly standard practice. Although in Fig. 4 no closer approach is possible, in other cases the wax could be thinned down so that the applicator actually touches the skin at the hot edge. In this case the hot edge dose is increased by 2 to 4 %, depending on the arrangement concerned, i. e. by considerably less than the difference

Table 2

Alternative arrangements for treatment of a patient with 6×8 cm wedge pair plus 8×6 cm normal field (see Fig. 4)

θ	s (cm)	N	Tumour dose %	Plateau depth (cm)	Depth below skin (cm)	H. S. factor
60°	6	25	118	1.7—9.0	5.7	1.45
60°	6	50	135	1.6—8.4	5.1	1.29
70°	5	25	121	1.5—8.9	5.9	1.42
70°	5	50	140	1.4—8.0	5.0	1.25
80°	4.2	25	122	1.4—8.1	5.4	1.35
80°	4.2	50	140	1.4—6.4	3.7	1.18
90°	3.5	0	105	1.7—8.4	6.1	1.52
90°	3.5	25	122	1.4—6.5	4.2	1.31

in dose between the surface and 1 cm deep at the hot edge for the individual wedge field. Moreover, it must be remembered that by eliminating the 1 cm wax thickness it is possible to decrease the separation, s, for the same value of θ, or alternatively, to decrease θ for the same value of s. The former does not necessarily give a net gain (see Diagrams 7 to 9) but the latter almost invariably does. For example, in an arrangement involving $\theta = 70°$, $s = 6$ cm, elimination of the 1 cm wax would enable the angle to be decreased to 50°. Table 1 indicates that in the former case, for $N = 50$, a plateau depth of 7.2 cm and hot-spot factor of 1.30 is obtained. In the latter case the plateau depth is 8.0 cm while the stated hot-spot factor of 1.23 is increased to 1.26 by the elimination of the wax. It is clearly advantageous to allow the wedges to approach the body as closely as possible.

Acknowledgements

The author wishes to record his thanks to his former colleague, Mr. K. E. Williams, who assisted in the plotting of the several hundred 'plateau curves' on which the analysis is based, and to Dr. L. A. W. Kemp for valued advice and discussion.

SUMMARY

The essential features of the dose distribution in a two- or three-field wedge arrangement may be extracted in the form of: mean percentage tumour dose, location of a defined 'plateau' relative to a base-line, and 'hot-spot factor', i. e. maximum skin dose/mean tumour dose. The following variables are studied: angle between wedges, separation of wedges, width of the normal field, width of the wedge field, and the slope and shape of the wedge isodose lines. The results of the analyses can be presented as 'dosage tables' which facilitate a rapid choice of configuration in individual cases.

ZUSAMMENFASSUNG

Die Eigenschaften der Dosisverteilung in einem Zwei- oder Dreifeldkeil können durch die mittlere Tumordosis, Lokalisation des 'Plateaus' relativ zur Basislinie und den 'hot-spot factor', d. h. das Maximum des Verhältnisses Hautdosis/mittlere Tumordosis dargestellt werden. Die variierbaren Grössen, nämlich der Winkel zwischen den Keilen, die Breite des Keilfeldes und die Steigung und Form der Keil-Isodosenlinien werden untersucht. Das Resultat dieser Untersuchungen kann als 'Dosis Tafeln' präsentiert werden, welche eine schnelle Wahl geeigneter Aufstellungen in individuellen Fällen möglich macht.

RÉSUMÉ

Les caractéristiques principales de la distribution de dose par une technique à deux ou trois champs avec coin peuvent être résumées par un pourcentage de dose moyen à la tumeur, par la localisation d'un 'plateau' par rapport à une ligne de base et par un 'facteur de point chaud', c'est-à-dire dose peau maxima/dose tumeur moyenne. Les variables: angle entre les coins, séparation des coins, largeur des champs normaux, largeur du champ avec coin, et pente et forme des lignes isodoses avec coin ont été étudiées. Les résultats de ces analyses peuvent être présentés sous forme de 'tables de dosage' qui facilitent le choix rapide d'une technique pour chaque cas.

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